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MEDICOLEGAL EXPERIENCES UNDER THE OHIO CORONER'S SYSTEM

Kurt E. Landé, M.D.
and
Garrett Boone, M.D., Hamilton, Ohio

On Oct. 12, 1945, recodification of the laws concerning the duties of the county coroner in Ohio was made effective. This final step in a long-drawn-out legislative process aimed at modernization did away with many antiquated and outdated laws and replaced them with rules reflecting more advanced concepts of investigation of the cause of death under doubtful circumstances. While still retaining the traditional office and name of coroner, the revised Ohio code allows the development of a coroner's system that resembles the medical examiner's system enacted in the last few years in Maryland, Virginia, and Georgia. These were modeled after the medical examiner's offices that have functioned successfully in Massachusetts, New York City, and Essex County, N. J., for a long time.

In Ohio, the coroner must be a physician. He receives an annual salary based on the population figure of the county in which he serves, and he is elected to office either as a true party candidate or as an unopposed, nonpolitical candidate. Each county coroner acts independently since there is no central agency, as in Baltimore, that coordinates and scrutinizes reports and that supervises the county medical examiners.[1]

Under sections 28855-5 and 28855-6 of the Ohio code, the duties of the coroner have been expanded and strengthened by the provision that "when a person shall die as a result of criminal or other violent means, or by casualty or by suicide, or suddenly when in apparent good health, or in any suspicious or unusual manner, the physician called in attendance or any other person finding the body or acquiring first knowledge of such death, shall immediately notify the office of the coroner and submit any information in his possession which may be requested by the coroner. . . . In such cases, if a request for cremation is made, the funeral director called in attendance, shall notify the coroner immediately. The coroner or his deputy, if he deems it necessary, shall take care of the body and may authorize an autopsy. The coroner may conduct an inquiry, subpoena witnesses etc. and shall make a report to the county prosecutor. The coroner or the county prosecutor may order a body disinterred for exhumation." The law permits the establishment of suitable offices, laboratories, and equipment in the county for the use of the coroner, and it allows the coroner to appoint and to fix the salaries of assistant coroners, one to be designated chief deputy coroner, and pathologists and other personnel to assist him in his work. It allows coroner's laboratories to perform examinations for coroners in counties that do not have laboratory facilities.

COUNTY SURVEYED

The survey of the practical working of the new coroner's laws was undertaken in an Ohio county with a population of 147,203 according to the 1950 census. Two industrial cities, Hamilton and Middletown, contribute 60,000 and 35,000 members of the population respectively. Rich farmland, a number of smaller communities, and one college town, Oxford, determine the semirural character of the county. About 3.5% of the population are Negroes who live in the two cities and in the college town. The turnover of population is considerable, because of constant interchange of the common labor force between southern Ohio and the hills of Kentucky and because of the usual transfer of personnel in industry.

The new coroner replaced an elderly physician who had been in office, under the old, antiquated system, for about 20 years. After the new coroner had been in office for some time, the effect of the new code could be evaluated, and it was only after his election that sufficient records were available to permit valid conclusions on the extent of work accomplished.

RESULTS OF SURVEY

Since the center of the county's population is in and around Hamilton, the greater number of postmortem examinations were handled in that city. The two Hamilton hospitals have a combined bed capacity of 415 as compared with a capacity of 148 in the Middletown hospital; therefore, proportionally greater numbers of patients who became coroner's cases were treated in Hamilton than in the neighboring city (table 1). The total number

1. Landé, K. E., and Wells, S. R.: Experiences Under the Medical Examiner System, J. A. M. A. **125**: 897-900 (July 29) 1944.

of deaths in the county amounted to 1,405 in 1951 and 1,440 in 1952. The total number of cases investigated by the coroner, 305 in 1951 and 334 in 1952, are about 21 to 22% of all the deaths in the county; this is a desirable optimum of investigatory activity. About every fifth death was reported to the coroner and investigated. In the majority of instances, a telephone call to the attending physician or to the hospital authority in whose institution the patient died, either from accident or shortly after admission, was sufficient to preclude any further steps on the coroner's part. Nevertheless, the number of cases, not quite one per day, shows that, in a county of about 150,000 inhabitants, the coroner's office has ceased being a sinecure; even if a case is closed after only cursory investigation, a complete record must be made and kept on file.

The five year total of 190 autopsies amounts to 13.1% of the coroner's cases. In 1951 and 1952 alone, the percentage was 15.7. Several factors have influenced the gradual increase of postmortem examinations. First, only since 1949 has a part-time pathologist been available at the Middletown hospital, and only since early 1950 has a full-time pathologist been available. Secondly, Mercy Hospital in Hamilton has been approved by the American Medical Association for intern and resident training and has, therefore, been more interested in raising its autopsy rate. For that reason, more coroner's cases have been autopsied at that hospital than the one in Middletown that does not employ medical house officers (table 2). In Hamilton, physicians have asked for and the coroner has consented to authorizations for autopsy in cases of traffic accidents, sudden death from natural causes, and other pertinent causes that perhaps would not have been subjected to examination if the hospital and house staff had not been interested. Furthermore, starting on Jan. 1, 1950, the coroner's pathologists in Middletown and Hamilton have been retained on a salary. This gives the coroner freedom to order autopsies whenever he considers them necessary, whereas formerly it was difficult to elicit payment from county authorities for the autopsies, even at the very low statutory Ohio rate of $20 per case (including written report and microscopic examination). Formerly the majority of coroner's autopsies requested for patients dying in one of the hospitals were not charged for in order to avoid any potential argument on the part of the county commissioners as to the need for such postmortem examination. It was felt by both the coroner and the pathologist that the development of an efficient coroner's service is a long-range program and that only gradual education of the public and its political representatives would and could lead to steady improvement.

TABLE 1.—*Coroner's Cases in Butler County, Ohio, from 1948 to 1952*

Year	Fatal Car and Railroad Accidents	Other Fatal Accidents	Suicides	Homicides	Death from Natural Causes	Rape and Abortion	Poison	Cases Investigated	Autopsies		
									Hamilton	Middletown	Total
1948	37	23	6	3	140	..	1	210	18	..	18
1949	40	25	12	5	184	..	..	266	24	5	29
1950	60	41	6	3	218	..	3	331	25	17	42
1951	49	34	16	2	203	..	1	305	39	12	51
1952	49	50	17	3	215	..	..	334	34	16	50
Total	235	173	57	16	960	..	5	1,446	140	50	190

From this point of view, the annual salary of $500 for the Middletown and $700 for the Hamilton pathologist was a step in the desired direction. Even so, one cannot claim that the money spent by the county for coroner's autopsies, listed in the county budget under the heading county morgue, represents a fair compensation for time and work involved in more than 30 autopsies per year performed by the Hamilton pathologist and about 15 by the Middletown pathologist. In 1952, the coroner received $2,233.05 for his own services and $1,466.08 for clerical and other expenses. Since the coroner's tasks often necessitate prolonged absence from his medical practice, it is not surprising that smaller counties in Ohio have had difficulty finding a physician willing to assume the duties of the coroner's office. It is not only the low pay offered that makes a physician think twice before he puts public interest before his own personal advantage but also the knowledge that for less vital but politically more important county offices rather lavish sums are available. This is especially true when one learns that in 1952, an election year, the county budget allowed only a total of $4,882.41, including the pathologists' salaries, to the coroner's office but $14,571.91 to the county dog catcher. If, under the present set-up, public-minded physicians take on the coroner's duties in the smaller counties, it is mainly with the realization that the slow process of educating the public to the practical importance of a well-functioning, up-to-date coroner's office has to be undertaken by some

TABLE 2.—*Coroner's Autopsies in Butler County, Ohio, from 1948 to 1952*

Year	Fatal Car and Railroad Accidents		Other Fatal Accidents		Suicides		Homicides		Death from Natural Causes		Rape, Abortions		Poisonings		Autopsies		
	H*	M†	H	M	H	M	H	M	H	M	H	M	H	M	H	M	Total
1948	3	..	1	..	2	..	1	..	11	..	..	..	..	..	18	..	18
1949	4	..	1	..	2	..	1	..	16	5	..	..	..	..	24	5	29
1950	2	1	1	1	1	..	1	..	20	15	..	..	..	..	25	17	42
1951	6	4	4	2	1	..	1	1	27	5	..	..	..	..	39	12	51
1952	4	2	3	..	..	2	2	..	25	12	..	..	..	..	34	16	50
Total	19	7	10	3	6	2	6	1	99	37	..	..	..	..	140	50	190

* Hamilton.
† Middletown.

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member of the medical profession. For years to come, those engaged in coroner's work will perform their task spurred more by their genuine interest in this fascinating field than by the opportunity to increase their personal income easily and conveniently.

CAUSES OF DEATH INVESTIGATED

The preponderance of coroner's cases were sudden deaths from natural causes. This is also the group in which the largest number of autopsies were performed; this incidence is similar to that of the whole country. Heart disease, mainly the direct or indirect sequelae of coronary arteriosclerosis, is by the far the leading cause of sudden, unexpected death. Degenerative diseases of the vascular system run a close second, with cerebral hemorrhages and ruptured congenital, cerebral, or arteriosclerotic aortic aneurysms frequently represented. Vascular accidents are so often brought to the coroner's attention because of their involvement in traumatic events at home, at work, or on the street. If an elderly person falls and contracts a fracture, quite often questions are raised concerning insurance, compensation, eventual liability of a third party, or justified or unjustified accusations of wilful or unintended neglect. Problems of this kind make an autopsy desirable, even if death occurs many months after the real or supposed accident took place.

Fatal hemorrhages of clinically unknown origin are another category encountered quite often; rupture of esophageal varices, hemorrhages from silent peptic or duodenal ulcers, and hemoptysis from unrecognized or neglected tuberculous cavities have been found. Sudden death from tuberculosis of any kind is not the only indication of the epidemiological importance of coroner's autopsies. Autopsies of children who died unexpectedly after attending a social function revealed pathological changes typical of Waterhouse-Friderichsen syndrome (fulminating meningococcic meningitis). Prompt prevention of its spread among contacts was initiated on the basis of the autopsy findings. The question of rabies came up twice, but the clinical suspicion was not confirmed anatomically. An upper respiratory infection that caused the death of two young brothers and clinically resembled malignant diphtheria but that was proved bacteriologically to have been caused by highly virulent hemolytic staphylococci prompted an extensive investigation by the county health authorities.

The new code fortunately provides that the coroner may proceed with postmortem examination in the instance of sudden death from suspected communicable or infectious diseases even if the family of the victim denies permission. Occasionally, the reluctance of the family to concede the value of an autopsy changes when the sudden death of one of its members is explained to eliminate any guilt feelings. This was dramatically demonstrated when it was proved that a 5-year-old boy, who died shortly after his father had given him a mild spanking, had suffocated from a malignant thymoma filling the whole mediastinum and compressing the trachea and esophagus almost completely.

Deaths caused by anesthesia are very tragic particularly when they occur in cases of elective or minor surgery. The coroner orders autopsies in most of these instances in order to rule out any other cause of sudden death and to establish anatomic facts in case of later unfounded accusations. In the case of a young woman who died before a thyroidectomy was begun, autopsy revealed unsuspected bilateral, huge adrenal cortical carcinomas. It is in cases of this kind that the Ohio code is unique in granting the coroner the undisputed right to have an autopsy performed.

Accidents are the next most important group of coroner's cases, with automobile accidents the commonest. Railroad accidents, however, are, in this county, far from rare and are frequently the result of unprotected railroad crossings at which drivers or pedestrians either half-asleep and/or intoxicated are caught by a train. Industrial accidents are encountered in the usual frequency, in spite of existing safety programs in the steel and paper mills, machine tool factories, and foundries. In the accident group, farm accidents, caused by machinery, livestock, or a combination of both, comprise a significant number. Among the industrial accidents, a few cases of poisoning have occurred; persons overcome by poisonous fumes while cleaning boilers have fallen into vats filled with concentrated acids or alkali solutions. In one case, an alcoholic derelict was exposed, while in temporary jail custody, to great quantities of delousing fluid. The police did not realize that it was dangerous for this man, who happened to suffer from a chronic, weeping generalized eczema, to absorb the fluid. Death resulting from accidental ingestion of carbon tetrachloride mistaken by a woman for a headache medicament while she was mildly intoxicated was misinterpreted clinically as happens so often when alcoholism is the well-known and obviously leading symptom. Two excellent discussions of accidental poisoning have been published recently.[2]

Although true homicides have been infrequent, suicides are fairly common; among them have been several homicide-suicide situations. Too many sleeping pills, gunshot wounds, and carbon monoxide are employed more often than hanging. Rape and abortion cases with fatal outcome have not numbered in the coroner's material since 1948. One case of death caused by septic abortion came to autopsy in 1945 and one in 1947. This infrequency does not mean that the county does not have its full quota of spontaneous and criminally induced abortions, but that those who start or perform them are apparently familiar with a certain degree of aseptic technique or have been lucky so far.

THOSE INTERESTED IN AUTOPSY RESULTS

The postmortem examination of many of the coroner's cases is very often of considerable interest to insurance carriers. For instance, a death originally reported to the coroner as accidental was conclusively proved to have been suicidal through the coroner's persistent investigative efforts. In spite of the obvious advantages to insurance carriers from an ever-expanding autopsy program, encouragement from that group has been singularly lacking. One often has the feeling that much future grief and litigation could be averted if the respective insurance carriers would show as much interest in having autopsies performed routinely as they demonstrate when they find

2. Moore, M.; Alexander, L., and Ipsen, J., Jr.: Death from Poisoning: Massachusetts, 1938-1948, New England J. Med. **246**: 46-52 (Jan. 10) 1952. Accidental Poisoning, editorial, ibid. **248**: 564-565 (March 26) 1953.

it particularly advantageous to utilize the coroner's findings. One sometimes wonders whether there could not be devised a procedure in which, in cases involving considerable amounts of money, those obviously profiting by the coroner's efforts could be made to share the expenses with the county for the coroner's work. Even without this financial consideration, one of the educational goals for which the coroner and his associates are striving is to change the passive attitude of these insurance companies to a more active, cooperative interest.

Without the close cooperation of the city police department, the sheriff's office, and the prosecuting attorney and his staff, no coroner could ever hope to perform useful work. They are by the same token the very groups who are cognizant of the help they receive from the coroner. In Butler County, as almost everywhere, these agencies and their representatives cooperate closely and to full mutual satisfaction with the coroner and his associates. A good deal of tact and diplomacy is needed, however, to prevent the coroner's being prevailed on and becoming the honorary but nevertheless very busy police and fire department attendant to patients who are alive and who have only a long-range relationship to the function of the coroner's office as outlined by law.

An alert physician serving as coroner and a pathologist serving under him, both of whom are well versed in forensic procedures, are indispensable in modern crime detection. Even in Butler County, enough cases occur in which only a thorough autopsy provides the basis for the further work of the police force that culminates in apprehension of a suspect and a later court trial. Or, in reverse, a postmortem examination may disclose, as in one Butler County case not long ago, that a woman beaten by her common-law husband did not die from the blows that were relatively minor in force but from a preexisting, extensive brain tumor. This finding closed the case as far as the law enforcement agencies were concerned and saved the county much unnecessary expense. Similarly in noncriminal cases only thorough and expert investigation can allow the decision of whether a man who, as a house painter, fell from a high scaffolding was the victim of a coronary thrombosis or whether a foundry worker who died at work suffered from an organic disease caused or aggravated by his occupation. Cases like these, which could be multiplied by numerous other random examples, belong in the coroner's field as much as do acts of pure violence, the investigation of which was the original basis for the task delegated to the coroner.

SUMMARY AND CONCLUSIONS

At present, as our five year survey shows, the coroner's office has the inherent opportunity for steady growth and organic expansion. For unusual circumstances like the eventual need for toxicological examination, laboratories such as those of the Kettering Institute in Cincinnati are available for Butler and other neighboring counties. Establishment of a centralized coroner's or medical examiner's system, similar to that of Maryland or Virginia, would appear justified and necessary only for those counties in which the service of a coroner's pathologist are not available. Since one of the main goals, in addition to steady and expanding routine work, is education of the general public on the local and county level as to the important role a well-functioning coroner's office plays in their welfare, it seems that for now the present Ohio system fulfills the existing needs and serves its purposes well.

116 Dayton St. (Dr. Landé).

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DIAGNOSTIC SIGNIFICANCE OF BLOOD SUGAR FINDINGS

Christopher J. McLoughlin, M.D., Lester M. Petrie, M.D.
and
Thomas E. Hodgins, M.D., Atlanta, Ga.

During the past few years the American Diabetes Association has sponsored a Diabetes Detection Week throughout the entire country. Many screening tests have been devised for finding the unknown diabetic, and a tremendous amount of work has been done on diabetes detection. The late Dr. James Edgar Paullin, in 1949, challenged the Georgia Department of Public Health to incorporate a test for abnormal carbohydrate metabolism in their Voluntary Multiphasic Health Test program, which included a chest roentgenogram, a test for anemia, and a serologic test for syphilis. The Anthrone method of blood sugar analysis was developed,[1] and, in 1950, the rapid screening of large numbers of patients was begun.[2]

Since April, 1950, about 600,000 persons in the state of Georgia have voluntarily submitted to this screening procedure for abnormal carbohydrate metabolism. A report on almost 250,000 of those screened during the past 18 months was made at a recent meeting.[3] Each person tested was classified as having an abnormal (over 160 mg. per 100 cc. in two hours or over 160 mg. per 100 cc. at 45 minutes, but below 160 at two hours) or a normal (below 160 mg. per 100 cc. both at 45 minutes and at two hours) blood sugar metabolism on the basis of an initial screening test and a modified glucose tolerance test. In the last group reported, 4,524, or 1.87%, were referred to their private physicians as cases of abnormal carbohydrate metabolism. To obtain a clinical follow-up on all referred persons, a mimeographed sheet (reproduced below) containing a check list of known causes of

From the Department of Medicine, Emory University (Dr. McLoughlin), and the Georgia Department of Public Health (Drs. Petrie and Hodgins).

Read before the Section on Internal Medicine at the 102nd Annual Session of the American Medical Association, New York, June 2, 1953.

1. Fetz, R. H., and Petrie, L. M.: Anthrone Blood Sugar Method, Adapted to Diabetes Case Finding in Multiple Screening Program, Pub. Health Rep. **65**: 1709-1718 (Dec. 22) 1950.

2. Petrie, L. M.; Bowdin, C. D., and McLoughlin, C. J.: Voluntary Multiple Health Tests, J. A. M. A. **148**: 1022-1024 (March 22) 1952. McLoughlin, C. J.; Petrie, L. M., and Fetz, R. H.: Diabetes Detection in Georgia, J. M. A. Georgia **40**: 285-287 (July) 1951.

3. Petrie, L. M.; McLoughlin, C. J., and Hodgins, T. E.: Carbohydrate Metabolism in General Population of Georgia, read before the American College of Physicians, Atlantic City, N. J., April 15, 1953.

hyperglycemia was forwarded, with results of the screening tests, to the person's private physician. He was requested to mail the sheet back to the health department after making his diagnosis as to the probable cause of the abnormal carbohydrate metabolism.

Analysis of our reports (see figure) of these persons with abnormal carbohydrate metabolism as to age, sex, and race shows that prevalence of abnormal sugar metabolism increases markedly with the advance in age, with the highest incidence of 8.98% in the group of Negro women in the seventh decade. Note that there is a definitely higher percentage of abnormal carbohydrate metabolism in the Negro females and a lower prevalence in the Negro males. Over 70 years of age the incidence in Negroes decreases slightly, but in the white population there is a very definite increase.

Diagnostic Check List

	Date	Blood	Urine
Primary test	.....	.....	.....
45 Min. Tolerance Test	.....	.....	.....
2 Hour Tolerance Test	.....	.....	.....

Serial Number.....

For the Doctor Only

Please check below your diagnosis to explain the abnormal sugar metabolism of the patient whose serial number in the health survey is indicated above and return to the health department. A franked envelope is attached. Please double check the factor which you feel is responsible for the abnormal sugar metabolism. Place single check beside other factors which coexist.

( ) Diabetes Mellitus	( ) Arteriosclerosis	( ) Hyperthyroidism
( ) Hyperpituitarism	( ) Chronic Arthritis	( ) Acromegaly
( ) Pregnancy	( ) Apoplexy	( ) Ulcerative Colitis
( ) Gall Bladder Disease	( ) Hepatic Cirrhosis	( ) Old Age
( ) Prolonged Bed Rest	( ) Low Carbohydrate Diet	( ) Overweight

( ) Previous Repeated Insulin Administration in a Non-Diabetic Individual

( ) Other Specify

Please indicate laboratory procedures which you employed:

( ) Glucose Tolerance Test	( ) Fasting Blood Test
( ) Post-Prandial Blood Test	( ) Fasting Urine Test
( ) Post-Prandial Urine Test	( ) Other Specify

If your findings do not confirm any abnormality of sugar metabolism, please explain.

Date..... Signature....., M.D.

Address.....

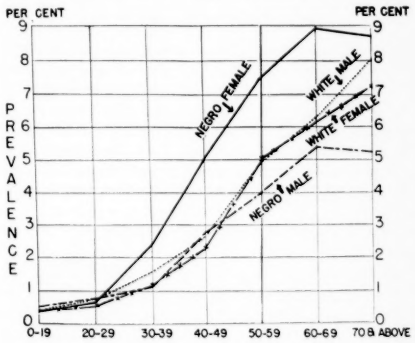
Revised Sept. 1951

Ga. Dept. of Public Health in cooperation with U.S.P.H.S.

There were 241,451 persons examined of whom 4,524, or 1.87%, showed some abnormality of carbohydrate metabolism and were referred to the physicians of their choice. In this group, 415, or 9%, gave a history of having known of a diabetic condition prior to taking the tests. This left 4,109 patients referred to physicians for further diagnosis. Of these, follow-up reports were submitted for 1,981, or 48.2%. We were interested in discovering what percentage of these patients referred would be diagnosed as having true cases of diabetes. Secondarily, we were interested in the various methods used by practicing physicians to determine the presence of diabetes. As we will show, a great deal of missionary work is yet to be done in training the average physician to make a diagnosis of diabetes mellitus.

Of the 913 patients classified by us as having borderline cases, 186, or 20.4%, were diagnosed by their physicians are being "new" diabetics. We classified 1,068 as having definitely abnormal tolerance curves by our screening tests, and 629, or 59%, were diagnosed as "new" diabetics. This gives a total of 815 new diabetics among the 1,981 reported. As will be demonstrated, however, there are, in our opinion, many cases of diabetes among the remaining 1,166 so-called "non-diabetics."

Among the laboratory procedures employed by physicians to substantiate or deny the presence of diabetes



Prevalence of abnormal sugar metabolism in 10 year age groups.

were (table 1) urinalyses, fasting blood sugar tests, postprandial blood sugar tests, glucose tolerance tests, including Exton-Rose tests, and other tests such as that for basal metabolism. The Folin-Wu method was used almost universally for blood sugar determination. No intravenous glucose tolerance tests or insulin tolerance tests were reported. Here, particularly, we would like to call attention to the fact that simple urinalysis or a fasting blood sugar test was used to substantiate or deny the presence of diabetes in more than 76% of all cases. In fact, more than 82% of the referred persons classified as nondiabetic by physicians were assured of the absence of this disease on the basis of no laboratory work at all, or nothing more reliable than a urinalysis or a fasting

TABLE 1.—*Laboratory Procedures Reported by Physicians*

Total 1,981	"New" Diabetic 815 (41.2%)	"Nondiabetic" 1,166 (58.8%)
Physician's Primary Laboratory Procedure:	No. of Patients "New" Diabetic	No. of Patients "Nondiabetic"
Urinalysis	419 (51.5%)	698 (60.0%)
Fasting blood sugar	210 (25.8%)	182 (15.6%)
Postprandial blood sugar	19 (2.3%)	21 (1.8%)
Glucose tolerance	161 (19.8%)	191 (16.4%)
Other *	6 (0.7%)	74 (6.3%)

* Usually history and physical. Three patients had basal metabolism determinations.

blood sugar test. It should be remembered that some of these referred patients had more than 400 mg. of blood sugar per 100 cc. on our screening or glucose tolerance tests.

According to the check lists submitted by physicians (table 2), among the 815 patients given diagnoses of diabetes, 178 were overweight, 104 had associated arteriosclerosis, and 75 were in the old age group (which

coincides rather closely with the percentage of diabetics previously reported [3] in the older age groups). Among these 815 new diabetics, 274 had one or more diagnoses other than diabetes checked by their physicians.

Among the 1,166 persons classified by physicians as having a condition other than diabetes as a primary reason for the abnormal blood sugar metabolism, 296 were overweight, 184 had arteriosclerosis, 108 were in the older age group, and 115 had their abnormal survey test explained as being due to a low carbohydrate diet. In this nondiabetic group of 1,166 cases, 25% had three or more diagnoses; 72 patients were checked as having five or more concomitant diseases and 125 as having other diseases, such as hypothyroidism, nephritis, chronic constipation, and low renal threshold, or as having a condition due to ingestion of too many cola drinks or too many starches. Even digestive glycosuria was mentioned as a cause of an abnormal two hour blood sugar level. There were 443 diagnosed as having no disease, although only six glucose tolerance tests were run on this entire

TABLE 2.—*Concomitant Diagnoses Reported by Physicians*

	"New" Diabetic	"Nondiabetic"
Overweight	178	296
Arteriosclerosis	104	184
Old age	75	108
Gallbladder disease	40	58
Arthritis	20	75
Low carbohydrate diet	17	115
Hepatic cirrhosis	11	34
Hyperpituitarism	3	8
Hyperthyroidism	3	36
Pregnancy	5	16
Ulcerative colitis	1	5
Prolonged bed rest	0	13
Apoplexy	2	5
Acromegaly	0	3
Other	28	125
No disease	0	443
Total	815	1,166

group; no other postprandial blood test was made; 162 fasting blood sugar determinations were made, and the remaining 275 had diabetes ruled out by means of urinalysis and so-called "clinical judgment." The diagnosis of no disease was made on the 162 tested with fasting blood sugars, despite the fact that in 42 cases the fasting blood sugar was over 170 mg. per 100 cc.

COMMENT

As a result of the screening tests for diabetes, only 41.2% of those referred to physicians as having an abnormal carbohydrate metabolism were diagnosed by their physicians as having true diabetes. The greater percentage of referred patients were considered nondiabetic by a series of tests, which, from every standpoint, were inadequate to rule out this condition. Therefore, in view of the apparent dependence of the practicing physician on thoroughly unreliable tests, we feel that further instructions in the proper method of diagnosis of diabetes mellitus should be included in any information sent to physicians when patients are referred. We must agree with McCombs and Finn [4] that the education of the physician is essential if we are to take advantage of the benefits of the case finding surveys. Even though only 815 of these patients were confirmed by their physicians' reports to us as being diabetic, we feel that these screening surveys are worth while. We believe that many of those diagnosed, or in our opinion misdiagnosed, as nondiabetic will in a year or so prove to be suffering from this disease.

4. McCombs, R. P., and Finn, J. J., Jr.: Group Health Surveys in Diagnostic Center, New England J. Med. **248**: 165-170 (Jan. 29) 1953.

SUMMARY AND CONCLUSIONS

We have demonstrated in Georgia an effective method for screening large cross sections of the population on a voluntary basis. It has been shown there is a definitely higher prevalence of abnormal carbohydrate metabolism in the Negro female and that in all older age groups the incidence appears to be much higher than previously anticipated. Too many physicians are still employing a fasting blood sugar test or urinalysis as a diagnostic procedure for diabetes. An educational program is being developed in Georgia to bring the physicians up-to-date on the diagnostic criteria of this disease.

1010 Medical Arts Bldg. (Dr. McLoughlin).

Symptoms of Potassium Deficit.—Potassium is the chief cation of muscle cells and of most other cells, since it is present in the *intra*cellular fluid. Sodium, on the other hand, is the chief cation of *extra*cellular fluids of the body. Any considerable replacement of sodium by potassium in the extracellular fluids is accompanied by serious disturbances and may be fatal. No other cation can entirely replace potassium in the intracellular fluid without interfering to a definite extent with the functional activity of the cell. . . . Potassium concentration of normal serum is 16 to 22 mg. per 100 cc., averaging 19 mg. While there is little or no sodium in the erythrocytes, the average concentration of potassium in them is about 420 mg. per 100 cc.

Absorption of potassium, under normal conditions, occurs almost solely from the gastrointestinal tract, but less than 10 per cent of potassium elimination occurs via the intestinal tract. Large quantities of it are so eliminated, however, in patients having diarrhea, but this excess may be due, in large part, to failure of reabsorption of digestive fluids rather than entirely to failure of absorption of the ingested salts. About 90 per cent of excreted potassium is eliminated in the urine. . . . The usual symptoms are general weakness, lassitude, diffuse atonicity of the intestinal tract with ileus, and cardiac irregularities, and hypotension. The severity of the symptoms and signs of potassium deficit are related to the serum potassium level. Decreased serum potassium effects neuromuscular physiology, the cardiac muscle, and the smooth muscle of the gastrointestinal tract.

All stages of change, from mere weakness to complete paralysis of the muscles of the trunk and extremities, may occur in the individual when the serum potassium level falls below 8 to 10 mg. per 100 cc., or 2.05 to 2.56 mEq. per liter. Changes in conduction, irritability, and contractility of cardiac muscle occur when there are potassium levels under 10 to 12 mg. per 100 cc. (2.56 to 3.07 mEq. per liter). These changes are manifested by abnormalities in the electrocardiogram. Clinically, the doctor may note systolic murmurs, gallop rhythms, cardiac dilatation, and hypotension. When potassium deficits are severe, cardiac arrest and death may occur.—Gail Gar Lyai Li, M.D., Hypopotassemia, as Encountered on a Gynecology Service, *American Journal of Obstetrics and Gynecology*, June, 1953.

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INFLUENCE OF PREGNANCY, LACTATION, GROWTH, AND AGING ON NUTRITIONAL PROCESSES

Tom D. Spies, M.D., Birmingham, Ala.

Disease is as old as living matter. From time eternal, man has sought means of easing his pain, relieving his disabilities, and discriminating between the foods that are good for him and those that are not. His progress has been made through a process of trial and error. It is the physician's duty to devise ways and means of aiding him. In this report I will discuss how the physician can best make use of his opportunity.

The cells of the human body, like all other living things, must adapt, grow, and reproduce. By the time one has become a mature adult, he has had perhaps thousands of illnesses, large and small, which he has overcome. If the investigator is to aid the practicing physician, the work they do must be integrated, dedicated to the truth, and applied toward preventing disease and maintaining health. In the present-day practice of medicine therapy, the nature of which little is known, is often applied; yet something may happen and relief may occur, temporary perhaps but relief nevertheless. Of that something that happens even less is known than about the therapy that is applied. My definition of health, as it might fit into a working concept, is the accurate, harmonious, and constant integration of all the necessary biochemical factors in the body. The vitamin balance, the endocrine balance, the electrolyte balance, the control of the body temperature, the maintenance of the blood glucose level, and the supply of oxygen to the cells are a few of the important factors. A derangement of any of these or of any other biochemical system disturbs the harmony of the cells and results in ill health. Ill health may come from trauma or infection or even from the disturbance of mental processes; many physiological phenomena are associated with emotions. For example, anger may cause the blood pressure to rise, sadness may bring excessive lacrimation, and excitement may result in insensitivity to pain; therefore, the state of mind alters the body functions. Mind and body work together continuously and are mutually dependent. Health is a normal outcome of such a harmonious relationship.

The success of preventive medicine depends on early perception of any source of disharmony. By the time the physical signs characteristic of disease are observed, the tissues have been badly damaged. Hence, the physician should attempt to develop insight into the biochemical action within the cell so that he will have knowledge of living processes. Because the mechanism of action is unknown, he must not fall into the error of thinking it is unknowable. To initiate this concept is a formidable undertaking, but it is one that must be undertaken.

The problems first to be considered are what causes chemical deterioration and how can such deterioration be predicted chemically and be chemically rectified. At the present time many physicians tell their patients, in effect, "to be their age, to take it easy, to slow down, and to avoid the pace that kills." In fact, they advise them how to live with their strokes, with their coronary accidents, with their emotional depressions, and with their pains and agonies, and how to grow old gracefully. It would mean a great deal if middle-life deterioration could be predicted in advance and reversed before serious symptoms occur. If the physician could avoid putting patients in beds and could allow them to work, and if further chemical deterioration could be prevented and the deterioration already present could be reversed, hospitalization could be cut down enormously and many young executives, technical men, and laborers could be kept in production for many years to come. Heart disease and strokes are not sudden causes of death. They occur as a result of years of chemical deficiency. To prevent disaster, a chemical audit, a diagnosis of chemical deficiency, and a corrective nutritional and chemical regimen are needed.

In 1928 studies were being made from the point of view that a human being is a product of the air he breathes, the water he drinks, and the food he eats; day after day, year after year, the tissues of the human body are replaced by intricate combinations of oxygen, water, and food. Today this is a medical axiom, and physicians have gone a long way toward showing that disease is chemical and chemically correctable.

DEFICIENCY DISEASES

After eight years of studying in the North, my associates and I initiated a study of deficiency diseases in an area in which they were endemic. At first we studied only adults with nutritive failure, but we soon learned that the children ate inadequate diets similar to those eaten by adults and that deficiency disease lesions, which waxed and waned with the seasons, developed in these children. At this time we observed that clinical evidence of deficiency diseases in adults was much commoner in women than in men, particularly in women of child bearing age. Our studies showed that the mother's diet often was inadequate during pregnancy and lactation, and frequently her supply of breast milk was not sufficient for the infant's needs. The infants were given foods from the family table, usually corn bread, syrup, biscuits, and grits, but few ever received orange juice or cod liver oil. These children were puny and fractious. They cried a great deal, were restless, and seemed too tired to play. A remarkable difference was observed between the personalities of these children with nutritive failure and those of normal children. Unlike normal children, they were apathetic and lacked the usual childhood interests.

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From the Department of Nutrition and Metabolism, Northwestern University Medical School, Chicago, and the Nutrition Clinic, Hillman Hospital, Birmingham, Ala.

The scientific evidence on which this report is based was produced by the author and more than 100 associates. The investigations described here were initiated 25 years ago and have been aided by many philanthropic persons, foundations, and corporations.

The researches on milk in the recent past have been supported by the National Dairy Products Corporation, the researches on rehabilitation by means of hormones by the Clayton Foundation for Research, and the researches on certain aspects of geriatrics by Hoffmann-La Roche, Inc.

Vitamin Supplementation.—We decided to ascertain what would happen when we administered synthetic vitamin supplements consisting of nicotinamide, thiamine, and riboflavin to these children. We soon learned that the lesions of pellagra, beriberi, and riboflavin deficiency disappeared following the administration of sufficient doses of these vitamins. The children's appetites tended to increase, and they gained in body weight. Unless their diets improved, however, the gain soon leveled off. It was obvious that the synthetic vitamins did not compensate for the deficiency of the other nutrients in their generally inadequate diets.[1]

By this time it was apparent that we should study the effect of chronic nutritive failure on the growth progress of children. During the past 15 years the growth patterns of over 3,000 children with and without nutritive failure have been investigated. In association with Dr. Samuel Dreizen, Miss Catherine Currie, and Miss Jo Gilley, we selected and repeatedly studied 2,000 children without evidence of nutritive failure and 600 children with chronic nutritive failure who had never received vitamin or other dietary supplementation.[2] All the children were seen frequently in the Nutrition Clinic, and on each visit height and weight measurements were made. The ages of these children ranged from 3 years to 16 years. The growth patterns were evaluated independently by the Wetzel grid technique and by a compilation of height and weight curves.

Analyses showed that, in children with nutritive failure, a substantial lag in height and in weight was present by the third year of life. Thereafter, the weight lag increased progressively in both boys and girls with nutritive failure. The height lag in boys with nutritive failure became progressively greater, while that of the girls remained fairly constant. The maximum mean isodevelopmental level [3] reached by boys with nutritive failure was 140 at 15 years 6 months. This level was reached by boys without nutritive failure 2 years 3 months earlier, when they were only 13 years 3 months old. The maximum mean isodevelopmental level reached by girls with nutritive failure was 130 at 15 years 3 months. This level was reached by girls without nutritive failure 2 years 6 months earlier, when they were only 12 years 9 months old. By the time the boys and girls with nutritive failure began to approach physical maturity, they had undergone a mean growth lag of over two years.

Milk Solid Supplementation.—The extent to which this retardation is reversible by means of dietary supplementation with milk solids has been investigated with regard both to over-all body size and to skeletal maturity. Eighty-two children with nutritive failure were paired by sex, skeletal age, grid position, and prevailing phase of growth. These children ranged in age from 4 years to 15 years. One child in each pair received a dietary supplement of milk solids equivalent in protein value to one quart of milk daily, six days per week, for a period of 20 months. No supplementation was offered the other child of each pair. By using the Wetzel grid technique, we found that 73% of the children receiving the supplement made more than one isodevelopmental level per month, as compared with only 39% of those in the control group (those not receiving the supplement). Improvement in physique was observed in 34% of the test subjects and in only 15% of the controls. Speed of growth was increased in 54% of the test subjects and in only 7% of the controls. Improvement in growth quality (indicated by improvement in physique plus an increase in speed of growth) was observed in 27% of the test subjects but in none of the controls.

In association with Dr. Dreizen, Dr. Richard Snodgrasse, Dr. George S. Parker, and Miss Currie, we used similar criteria in selecting an additional 41 pairs of children. These children ranged in age from 1 year to 10½ years. The children in the test group were given milk solids equivalent in protein value to three quarts of milk a week for a period of 40 months, while the control group did not receive a dietary supplement of any kind.[4] Although the test subjects in this group received the same total amount of milk solids as those in the first group, the milk solids were given to the second group over a period of time twice as long, so that the average weekly amount was only half of that given to the first group. At the end of the 40 month period no differences between the test and control groups were measurable either by the Wetzel grid technique or by the skeletal maturation techniques, thus showing that we had arbitrarily selected an average weekly amount of milk that was too small to produce measurable changes.

In 19 of the test subjects the amount of milk solids was increased to the equivalent of 12 quarts of milk weekly (2 quarts a day, six days a week). This was four times the average weekly amount given during the 40 month period and twice the average weekly amount given during the 20 month period. The remaining test subjects continued to receive the three quart weekly level. To facilitate giving 12 quarts of milk weekly and to make it possible for the entire daily amount to be given in one feeding, one-half the milk solids was reconstituted in as small an amount of water as possible and frozen to the consistency of ice cream. One-half ounce of sugar and a few lemon drop chips were added for flavor and palatability. The other half of the milk solids was reconstituted to either a pint or half-pint volume (depending on the age of the child) and fed with the ice cream. This supplement was fed for a period of six months. At the end of the six month period the rate of skeletal maturation in the test subjects had increased by 80%. The increase in skeletal maturation of boys receiving the 12 quart weekly level exceeded that of their paired controls by 93% and exceeded that of boys receiving the 3 quart weekly level by 65%. The increase in skeletal maturation of girls receiving the 12 quart weekly level exceeded that of their paired controls by 90% and exceeded that of girls receiving the 3 quart weekly level by 90%. In 17 of the 19 test subjects (89%) on the equivalent of 12 quarts of milk weekly, the rate of skeletal maturation in the most advanced centers increased, thereby reducing the lag between their actual and genetically limited growth rate. This occurred in only 45% of the children receiving the

1. Spies, T. D.; Ewing, G. D., and Mann, A. W.: Effect of Synthetic Niacin Amide, Synthetic Thiamine and Sythetic Riboflavin on Infants and Young Children with Deficiency Diseases, Arch. Pediat. **61**: 517 (Oct.) 1944.

2. Spies, T. D.; Dreizen, S.; Currie, C., and Gilley, E. J.: Unpublished data.

3. By the Wetzel grid technique, isodevelopmental level is a measure of the body surface area based on the height and weight of the child.

4. Spies, T. D., and others: Unpublished data.

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three quart weekly level and in 46% of the controls. Acceleration in the rate of maturation of the least advanced bone center was shown by 58% of the children receiving the 12 quart weekly level and in only 23% and 27%, respectively, of those receiving the 3 quart weekly level and those serving as controls.

Despite differences in nutritional requirements at different ages, there appears to be no relationship between chronological age and the degree of increase in bone maturation. From this study it appears that the removal of deficiencies of skeletal maturation in the child, regardless of age, is dependent primarily on the adequacy of the level of supplementation, thus the retarding effect of prolonged nutritive failure is reversed promptly if the nutrients in the dietary supplement are supplied in sufficient amounts to overcome the accumulated deficiencies. Brief summaries on the following two patients are illustrative:

CASE 1.—This girl, the youngest of five children, was born at a time when her family's economic status was at its lowest. Her father, who had worked as either a miner or a sharecropper since the age of 13 years, was in a mining accident two years prior to the child's birth and was never again able to work. From the time of his accident until his death eight years later, the family was dependent on charity. They received from $8.00 to $18.00 per month. Their diet consisted of dried beans, corn bread, biscuits, and occasionally a small amount of fat pork. During this interval pellagra developed in the mother and four of the children, and they were treated by us. When we first examined the girl, she was nervous, cross, and apprehensive. She cried frequently, did not sleep well, and complained of muscular cramps, headaches, and stomach-ache. She had bilateral cheilosis. She was a small child who looked like a wizened old lady. In school she had not done well; she had repeated each semester at least once and was only in the third grade at 12 years of age. She enjoyed playing with the children in her own grade, who were about four years younger than she.

The grid analysis for this girl is seen in figure 1. When she was 7 years 10 months of age, she had clinical pellagra and had an extremely poor physique (channel B-4 on the grid). She was progressing on a very slow age schedule. Her developmental age (using the 67% auxodrome, or age schedule, as the standard of reference) was 5 years 7 months, showing a growth lag of 2 years 3 months. When she was given nicotinic acid, the lesions of pellagra disappeared and she felt better than she had prior to this therapy. Her diet remained inadequate, however, and at the end of a year she was retarded 4 years 6 months in over-all body size. This indicated that, although the nicotinic acid was specific for nicotinic acid deficiency, her growth and development could not improve until her dietary intake improved. Her diet then became somewhat more adequate, and by the time she was 11 years 6 months of age, her growth lag was 2 years 10 months. We decided to put her on supplementary feedings of reconstituted dry milk solids, and she was given the equivalent in protein value of one quart of milk per day, six days a week, for a period of 20 months. No further improvement in physique occurred, but her speed of development continued to increase slowly. In the 20 month period her retardation in growth was reduced by five months. During the time she received the milk supplement, she was cheerful, friendly, and pleasant. Her mother and all other observers noted that she had more energy, slept better, and was less nervous than she ever had been. Her teachers saw a definite change in her ability to concentrate on her studies and to retain information that she acquired. They noted also that she had a great deal more energy, and for the first time she would participate in group activities. From that time on, she had no failing grades and was delighted and proud of herself when she passed to the fourth grade.

CASE 2.—The patient's family, as that of case 1, was in a state of nutritive failure from the time the members were first examined in our clinic. The patient, 1 of 11 children, was breast-fed until she was 18 months of age and had no supplements such as orange juice or cod liver oil. She was given butter bean soup "to start real eating." As a small girl, she was easily fatigued and had frequent headaches and severe cramps in her legs. Most of the time she fretted and cried, and frequently she had bilateral cheilosis. At best, her two meals a day included butter beans, salt pork, bread, and molasses. On several occasions treatment with riboflavin was followed by relief of the cheilosis. When she was 12 years of age, a supplement of dry milk solids reconstituted in water was initiated. Promptly she began to grow taller and to fill out. After she had received the milk supplement for 14 months, her body changed from that of a scrawny, thin girl to that of an adolescent. Her energy and appetite increased, and she had no lesions of riboflavin deficiency. When the therapy with a supplement was discontinued, the cheilosis returned. When she was treated again, the lesions disappeared.

The grid analysis from case 2 is shown in figure 1. As can be seen, when she first was seen in the clinic, her physique was fair (channel B-2) and she was progressing on a very slow age schedule (98% auxodrome). Using the 67% auxodrome as the standard of reference, she was retarded 1 year 6 months and her developmental age was 6 years 6 months. Further loss of physique occurred together with progressively greater re-

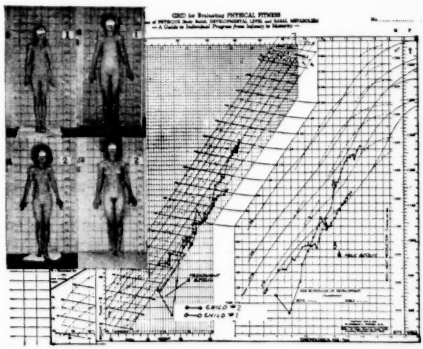

Fig. 1.—Wetzel grid analyses for case 1 and case 2.

tardation, until at the age of 10 years 4 months she was 2 years 8 months retarded. At that time she began to progress, and her speed of development (only one incident of growth failure, occurring at 11 years 4 months) increased, until at the age of 12 years she was retarded only 16 months. At this time she was placed on the dietary supplement of dry milk solids equivalent in protein value to one quart of milk per day, six days a week, for a period of 14 months. Throughout the period of supplementation, she continued to improve with less fluctuation and continued to increase her speed of growth. At the end of the period of supplementation she had a fair to good physique and was retarded only seven months. Her retardation was reduced by nine months in the 14 months of supplementary feeding. The differences in maturity, as indicated by secondary sex characteristics, are clearly evident in the photographs (fig. 1) of the two girls.

Solid Food Supplementation.—Nutritionally speaking, a 9-month-old baby has had 18 months of nutrition, that is, including the 9 month prenatal period. With birth the whole question of adjustment arises. How soon the baby can take solid foods is an important matter. The revolutionary schedule of giving solid foods early is one of recent vintage. For too long this type of food was not offered to the infant, since it was considered that a baby's stomach was not big enough to contain sufficient food to

provide adequate nourishment and that he should have frequent feedings in liquid form. Recently we began studying the introduction of food other than milk as early as the second day of life. For a special long-term study we selected 24 children, ranging in age from 2 months to 2½ years, and placed them on supplementary feedings of Swift's meats for babies. Children under 1 year of age

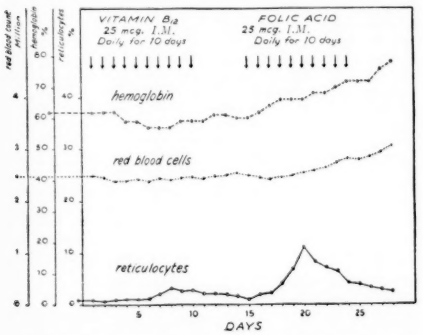

Fig. 2.—Comparative evaluation of vitamin B_{12} and folic acid per unit of weight in a patient with macrocytic anemia of pregnancy.

arbitrarily are given strained food, and those over 1 year of age are given chopped foods. Each subject receiving the meats has accepted this supplementation enthusiastically and appears to be growing at a faster and more regular rate than his control.

Rickets: In our clinic it is not uncommon to see an obvious case of rickets. It always has interested us that severe clinical rickets occurs not only in children who lack vitamin D or sunlight exposure but also in some children who grow rapidly. This rapid growth has a great deal to do with the clinical evidence of rickets. Clinical rickets, as we see it in Alabama, occurs chiefly among the Negro children who sleep a great deal during the day. Those who are out in the sun show no evidence of rickets. Occasionally one sees white children with rickets, but usually they are children who are growing very rapidly. The incidence of rickets is limited almost entirely to the temperate zones. The characteristic deformities are not so common now as formerly, but we still see the milder form. In some ways the mild form is more serious, since it is more difficult to recognize and, therefore, may escape treatment. It is well known that the pathogenesis of rickets lies in the condition of the calcium and the phosphorus in the blood serum and that in rickets the concentration of phosphatase in the serum is increased. Human milk is by no means antirachitic. To prevent rickets, vitamin D should be administered. Vitamin D produces its regulatory effect on the content of calcium and phosphorus in the fluids of the body. With satisfactory treatment the bony deformities of rickets gradually disappear, the bones acquire an increased degree of rigidity, and the muscles seem to improve.

Anemia: A medical mistake being made at the present time is failure to administer iron in cases of iron deficiency anemia. When the physician finds that the patient cannot tolerate large doses of iron because of the resulting gastritis or diarrhea, too often he gives the patient no iron at all. Smaller doses would be well tolerated and, if given over a long enough period of time, would produce satisfactory responses.

Ascorbic acid, folic acid, and vitamin B_{12} are nutrients that are essential for health and well-being. Vitamin B_{12} is essential for the integrity of the nervous system and the bone marrow. Folic acid is essential for relieving the macrocytic anemia of pregnancy and of infancy and is most useful in the treatment of persons with endemic sprue or nutritional macrocytic anemia. Figures 2 and 3 indicate our recent experiences and observations with these materials. The use of ascorbic acid in the treatment of scurvy is extremely well known. It, like pantothenic acid, seems to be essential for the integrity of the adrenal glands.

Obesity: The obese patient presents a medical problem common to most practicing physicians. Patients offer many excuses for overeating, but, if they will consider their problem realistically, they are inclined to agree with the diagnosis of obesity. What they really do not like about the diagnosis is the implication that they are "eating themselves fat." Many reducing diets are unbalanced and actually inadequate in a number of respects. Nonfat dry milk solids are being used more and more as a basis for reducing diets. Because they are an excellent source of protein and calcium, they make an important contribu-

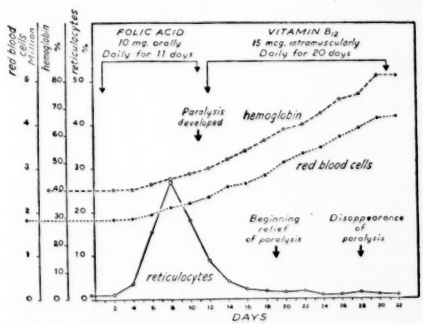

Fig. 3.—Development of symptoms of acute combined degeneration of the spinal cord in patient with pernicious anemia who was receiving folic acid therapy; the symptoms disappeared with vitamin B_{12} therapy.

tion to a reducing diet. A nonfat dry milk that we have found acceptable and useful is a product that has vitamins and minerals added in the following amounts per quart:

Vitamin A	4,000 USP units
Vitamin D	400 USP units
Vitamin B_1	1 mg.
Vitamin B_2 (riboflavin)	2 mg.
Niacin	10 mg.
Iron	10 mg.
Iodine [5]	0.10 mg.

Physicians are beginning to understand that overeating often is a manifestation of a fundamental disturbance that is primarily psychological or emotional in nature. Leonard Williams has said that obesity is divided into

5: Iodine may be added in the Midwest because of the incidence of endemic goiter.

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three stages known, respectively, as the enviable, the comical, and the pitiful. This is a false estimate of values. Obesity is always tragic. A list of its hazards is terrifying.

DISEASES IN OLD AGE

Every investigation of nutritive failure that we have made in the past 20 years has indicated the predominance of mixed, rather than single, deficiencies of elements essential for life. The margin of safety against certain deficiency diseases is narrow rather than broad. Figure 4 shows the age incidence of deficiency diseases in males and females. Note that during the child bearing period of life many more women are affected with clinical evidence of deficiency disease. After this period is over, this difference gradually disappears, and after the age of 70, the age incidence of dietary deficiency diseases seems remarkably similar in men and women. For a quarter of a century I have had a deep interest in trying to understand the underlying chemical changes that precede senescence. A long time ago we became impressed with the extreme variation in response to the wear and tear of life. It seemed very striking that some persons weathered the years from 45 to 70 in such a way as to appear to be still as biologically, physically, and mentally active as they were at the age of 40 and seemed destined to live a number of years. The true killers such as cerebral accidents, coronary disease, and cancer of the stomach, which strike so often between 45 and 70 years of age, seem less likely to occur after the age of 70

In this report it is not possible to mention all the combinations of vitamin and mineral deficiencies that might occur. As a part of our over-all concept, we consider that the nutrients essential to the growth and development of infants and children are necessary for the maintenance of vigor and strength of the person who has matured. We have chosen a number of older persons with well-recognized chronic syndromes and demonstrated that their health can be so improved that they can be returned to gainful occupations. How much better it would be if susceptibility tests were available that would tell in advance which persons should be observed and for what, so that truly scientific preventive medicine could be done. When that day comes, physicians shall be busy preventing disease rather than treating patients in late stages of disease.

Leukemia.—A number of substances are directed toward destroying existing leukemic tissue and toward preventing the multiplication of leukemic cells in persons with leukemia and lymphosarcoma. None of these substances is a positive cure for the disease. We have been concerned especially with the effect of corticotropin (ACTH) and cortisone and have seen some interesting results, particularly in cases where the bone marrow has been heavily infiltrated. The brief case history that follows indicates what can be accomplished in selected cases:

CASE 3.—A 53-year-old white woman was brought to the clinic because of loss of weight and strength, a breaking out of the skin, and swellings in her neck. She had been in good health until a few years before admission, when she noticed that she lost her strength insidiously and could not do her housework as well as usual. She eventually could not do her housework at all; soon a rash appeared all over the body. Swellings appeared under her arms and in her neck. Her white blood cell count was 78,000, and at this time she was given x-ray therapy. The white blood cell count was reduced to 14,000, and she felt somewhat better. After this, however, she promptly suffered a relapse, and x-ray therapy was found to be of no value. Six months after she had received no therapy whatsoever, she returned to the clinic. She said that she "just hardly could go." One hundred milligrams of pituitary corticotropin (Armour) was administered intramuscularly in divided doses every six hours. Promptly she began to feel better, her leukocyte count fell from 60,000 to 13,850, and hemoglobin rose from 36% (5.6 gm.) to 79% (12.2 gm.). Her weight increased 10 lb. (4.5 kg.), and she gained greatly in strength. After being treated for a month, she felt very well. She had started to do her own housework again. Therapy was discontinued at that time.

While examination reveals some swelling of the lymph nodes and blood smears show characteristic leukemia, the patient in case 3 has been free of symptoms for over three years. We do not understand the precise chemistry of what has happened in this patient or in others who have improved, nor do we understand the difference between the patients that improve and those in whom a satisfactory response does not occur. Perhaps any form of leukemia is not a single disease but is the result of a variety of causes. At any rate some vital reaction occurs.

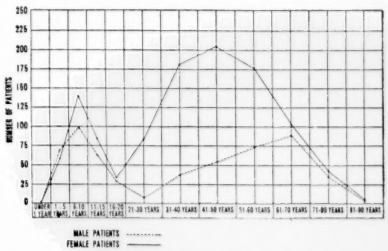

Fig. 4.—Age incidence of patients with deficiency diseases in 1944.

Since we have seen no patient who has returned entirely to normal, we must conclude that there is no known method of treatment that is a cure for leukemia.

Chemical Disturbances.—In our approach to the chemical disturbances that may affect persons of "advanced maturity," we keep in mind that these disturbances are responses of living tissues to abnormal chemical situations. We know that tumors existed in prehistoric animals, and in our study of the problem we have chosen many avenues of approach.

Several years ago Dr. Rolf Ladisch submitted to me a hypothesis that incorporated the concept that the quinones from certain beetles might be carcinogenic under certain circumstances. Dr. Ladisch, Dr. Dreizen, and I have worked with materials from the thoracic and abdominal glands of the Tribolium confusum and Tribolium castaneum species of beetles, insect infestants of common cereals, in order to observe their effect on living matter. From the secretions of these glands we have obtained and used three quinones with chemical structures resembling carcinogenic agents. To 36 rats of the Long-Evans strain, these materials were given in varying doses in the food, in the water, or by subcutaneous injection. For a long period of time there were no noticeable effects on the growth rate, health, or physical

appearance of the rats so treated. Eventually 10 of the 36 rats in the experimental group and 1 of the 9 litter mate controls (not given the test material) showed a progressive decline in weight, extending from four to six weeks prior to death. Gross and microscopic evidence of tumor formation was found in each of these animals. The structure of these tumors can be seen in figure 5. From these preliminary observations it would seem that the quinones isolated from the glandular secretion of the beetles probably provoked tumor formation and that the tumors are of the type described by Gillman and Gillman in their classic work on experimentally produced lymphomata in rats.[6] Many questions arise that eventually must be answered. Could these masses be transplanted? Why did tumors not develop in the majority of rats? Is there any practical importance to these observations, or do they merely ascertain the information that the administration of the quinones may be followed by tumor formation? At least these preliminary observations fit into our over-all concept that proper chemical environment is of utmost importance and is essential for the health of all living tissue.

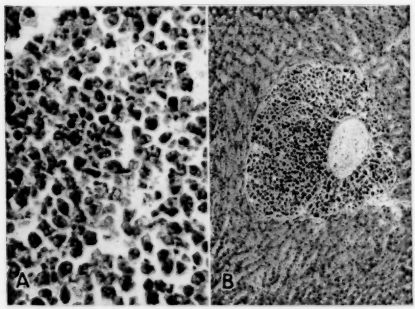

Fig. 5.—Tumors found in rats injected with carcinogenic quinones from beetles. *A*, abdominal mass (× 167), and *B*, liver showing normal liver pattern surrounding a well-demarcated cluster of abnormal cells (× 30).

Psychotic Reactions: For many years it has been known that pellagrins often manifest psychotic changes, but the association of psychoneurotic symptoms with subclinical and mild pellagra was not appreciated until it was pointed out that these patients were frequently disgnosed as neurastheniacs. We learned that these symptoms had certain distinctive characteristics, that they were significant in making an early diagnosis of the disease, and that such symptoms disappeared dramatically following nicotinic acid therapy. In addition to the elementary syndrome associated with it, there were the general symptoms of central nervous system involvement characterized by weakness, increased fatigability, sleeplessness, and headaches.

As a part of the elementary psychosensory disturbance, the patients described conditions as making them feel "uneasy" or as bothering or hurting them. They could not tolerate bright colors or bright lights, and noises such as the slamming of a door or the screaming of a child produced a great emotional shock, with the result that they felt "jerky and shaky." As the condition became severer, these noises became unbearable. They found that odors that had never bothered them before were so disagreeable that they caused nausea and vomiting. Several patients who had enjoyed cooking said that the orders become so unpleasant that they caused distress and agony. Tastes changed so that they disliked foods intensely. These patients were tense, irritable, and, as they said, "on edge." Prominent complaints were dizziness, a feeling of uncertainty, and difficulty in maintaining balance. They complained of feeling "restless on the inside" and of being much more excitable and sensitive than they were before their illness. Their emotional reactions were directed toward a depressive response; they had sad, gloomy moods associated with constant apprehension. They seemed constantly to be engaged in a struggle between a desire to rest and a feeling of restlessness. The breakdown of personality was striking: persons previously strong, courageous, and enduring became wary and apprehensive.

Pernicious Anemia: We have known for some time that a rare patient with pernicious anemia in relapse will respond to as little as 5 mcg. of vitamin B_{12} administered orally, whereas others will respond only when a dose of several milligrams is given. For many years we have been interested in the fact that patients with pernicious anemia lack a certain enzyme in their gastric juice. There is much current interest in intrinsic factor preparations, which act as activators of vitamin B_{12}, making it possible to decrease greatly the oral dose of this vitamin. Several pharmaceutical concerns have produced active intrinsic factor preparations that we have found will potentiate the effect of vitamin B_{12} administered by mouth to patients with pernicious anemia in relapse. The response of such a patient to this therapy is illustrated in the following case history:

Case 4.—A 72-year-old machinist was brought to the clinic in late 1948 complaining of weakness, fatigability, and occasional soreness of the mouth and tongue. Fatigability and loss of strength began about three years previously. He felt tired all the time and was content to just sit all day. The soreness of his mouth and tongue began two years previously, recurred intermittently, and varied in severity; at times he had difficulty in even drinking water. A sensation of numbness of his feet and of having pads of cotton under his toes had been present for a year and a half. Numbness of his fingers had been present for three weeks. For several weeks he had noticed dyspnea on exertion. A year and a half before he was admitted, he consulted a physician who told him that he had anemia bordering on pellagra and gave him an injection every other day for about a year. In the patient's opinion this therapy gave him little, if any, relief. Because his work required a high degree of skill in using his hands, he had not been able to work regularly.

Physical examination showed generalized pallor. The scleras and conjunctivas were pale, and there was slight icterus of the scleras. His tongue was slightly thickened, and there were scattered denuded papillae at the tip and along the margins. The findings of the neurological examination were within normal limits. An oral glucose tolerance test showed a normal curve. Repeated gastric analyses showed no free hydrochloric acid in the gastric juice even after histamine stimulation. The stools were normal. There was megaloblastic arrest of the bone marrow, characteristic of pernicious anemia in relapse.

6. Gillman, J.; Gillman, T.; Gilbert, C., and Spence, I.: Lymphomata (Including Hodgin's-Like Sarcomata): Their Experimental Production: A Study of Their Pathogenesis and Etiology and a Comparison with Corresponding Tumours in Man, Clin. Proc. **8**: 222 (Dec.) 1949; The Pathogenesis of Experimentally Produced Lymphomata in Rats (Including Hodgkin's-Like Sarcoma), Cancer **5**: 792 (July) 1952.

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The initial peripheral blood values were red blood cells 2,870,000, hemoglobin 72% (11.1 gm.), and reticulocytes 0.4%.

He was given a single 50 mcg. oral dose of vitamin B_{12}. His blood values and clinical condition remained essentially unchanged for a month. Then he was given a single 450 mcg. oral dose of vitamin B_{12}. Four and five days later his reticulocyte count was 4.8% and 5.0%, respectively, and subsequently there was a slight increase in red blood cells and in hemoglobin. His fatigue was relieved slightly, he felt better in general, and his color improved. Neurological examination at this time showed slight hyperesthesia of the distal third of the forearms, of the hands, and of the fingers, slightly severer in the fingers. There was slight hyperesthesia from just above the knees to the ankles. There was a tingling sensation of the legs and slight tenderness of the heels.

He was given a single 25 mcg. intramuscular injection of vitamin B_{12}. Four days later he volunteered that there was less tingling of his fingers and numbness of his toes and that he no longer had any burning sensation of his tongue. By the following day all the redness of his tongue had disappeared, and he said that he had more energy and that he "felt like doing something." The numbness of his feet and the tingling of his fingers continued to subside. Nine days after the first injection of vitamin B_{12}, he was given a second injection of 25 mcg. intramuscularly. Within the next three weeks the symptoms of nerve involvement, all the soreness of his mouth and tongue, and the dyspnea disappeared gradually. At the end of this time his blood values were red blood cells 4,210,000; hemoglobin 83% (12.7 gm.); and reticulocytes 1.8%. A month later he went back to work full time.

In the following four years he had intermittent therapy with vitamin B_{12}. In March, 1953, he complained of fatigue, tingling and numbness in his fingers, and a burning sensation of his tongue, which was moderately reddened on both upper and under surfaces. His blood values were erythrocytes 3,240,000; hemoglobin 73% (11.3 gm.); and reticulocytes 2.8%. He was given four tablets of vitamin B_{12} and an intrinsic factor concentrate daily for 10 days. On the fourth day of treatment he felt less fatigued and the soreness of his mouth decreased. The tingling and numbness of his fingers decreased on the sixth day of treatment. By the seventh day he said that he felt "like a 2-year-old." By this time all the redness and soreness of his tongue had disappeared, and by the ninth day he no longer had any sensation of numbness and tingling of his fingers. On the seventh day of therapy his reticulocyte count was 10.8%. On the 10th day his red blood cell count was 3,750,000 and his hemoglobin 83% (12.8 gm.). Three weeks after this therapy was initiated, his red blood cell count was 4,070,000 and his hemoglobin 72% (11.1 gm.).

After the first period of therapy in 1948, he began working regularly. His work requires a high degree of skill in the use of his hands. He measures with a micrometer, and measurements must be accurate to one-thousandth of an inch. He says, "I feel a thousand times better than I did when I first came to the clinic, and I am happy to work again."

Studies of this case and many others show that, in order to determine the intrinsic factor activity of any preparation, one must first do carefully controlled tests showing that the oral administration of the amount of vitamin B_{12} in the preparation cannot, in itself, produce any detectable effect. Then one must show that the administration of the same amount or less of vitamin B_{12} and a potent intrinsic factor concentrate is effective under the same conditions (fig. 6).[7] After years of testing, I prefer to conduct the tests using 5 mcg. of vitamin B_{12} per day, although we often use 10 or 15 mcg., or even more. The larger amounts are less satisfactory because they require more careful and definite control. We have found that patients who do not respond to 5 mcg. of vitamin B_{12} orally have a maximum hemopoietic and clinical response when as little as 1 mg. of the purest concentrate of intrinsic factor is administered along with 5 mcg. of vitamin B_{12}. We have given concentrated intrinsic factor and vitamin B_{12} as long as two hours apart, and a definite hemopoietic and clinical response has followed, indicating that the intrinsic factor and vitamin B_{12} must interact in the intestinal tract. The purest preparations of intrinsic factor concentrate show definite evidence of being a protein. Obviously such studies

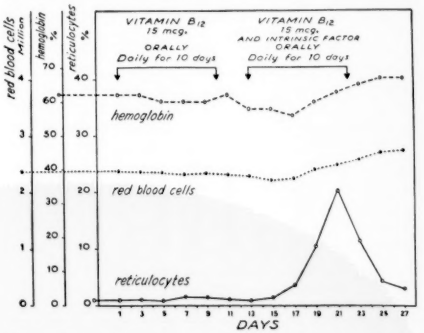

Fig. 6.—Hemopoietic response of patient with pernicious anemia to the oral administration of vitamin B_{12} alone and with intrinsic factor.

show that the day of practical oral therapy with vitamin B_{12} is near at hand for the physician who chooses to use it for selected patients.

In many cases vitamin B_{12}, like some of the other vitamins, has a special effect on appetite. This appetite stimulating property is not precisely related to the increase of red blood cells or hemoglobin in the blood. When patients with pernicious anemia or tropical sprue in relapse are given transfusions and their blood values increase, they do not get the remarkable increase in strength, vigor, and appetite as is induced after the ad-

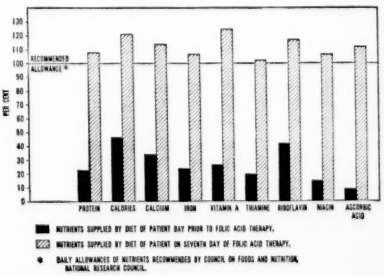
RECOMMENDED ALLOWANCE *
PER CENT
PROTEIN
CALORIES
CALCIUM
IRON
VITAMIN A
THIAMINE
RIBOFLAVIN
NIACIN
ASCORBIC ACID
NUTRIENTS SUPPLIED BY DIET OF PATIENT DAY PRIOR TO FOLIC ACID THERAPY.
NUTRIENTS SUPPLIED BY DIET OF PATIENT ON SEVENTH DAY OF FOLIC ACID THERAPY.
* DAILY ALLOWANCES OF NUTRIENTS RECOMMENDED BY COUNCIL ON FOODS AND NUTRITION, NATIONAL RESEARCH COUNCIL.

Fig. 7.—Nutrients supplied by diet of patient with pernicious anemia before and after folic acid therapy.

ministration of large amounts of vitamin B_{12} or folic acid (fig. 7 and 8). In studying selected patients with pernicious anemia we have learned that when their blood values increase to normal following repeated transfusions, they feel fine; if they are given injections of sterile sodium

7. Spies, T. D., and others: Antianemic Properties of Reaction Products of Vitamin B_{12} and the Intrinsic Factor, J. A. M. A. **151**: 1264 (April 11) 1953.

chloride, they do not get an increased sense of well-being; when vitamin B_{12} is administered without their knowing they are getting different injections, they do get a definite "lift" and feel much better.

Vitamin B_{12}, like folic acid, thiamine, nicotinamide, riboflavin, and ascorbic acid, is singularly free from unpleasant side-effects. It has a most important place in practical therapeutics. The evidence of 25 years of work shows a wide variation in the individual needs of patients

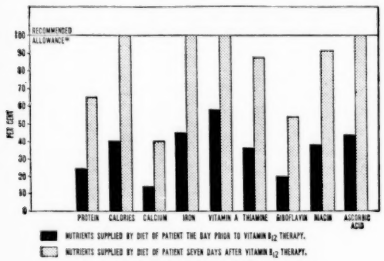

Fig. 8.—Nutrients supplied by diet of patient with tropical sprue before and after vitamin B_{12} therapy.

with pernicious anemia. The value of vitamin B_{12} as a growth factor for children cannot be regarded as established. One cannot, by physical examination or studies of the blood or bone marrow, tell before therapy whether or not a person has a deficiency of folic acid or vitamin B_{12}. Folic acid has a special relationship to pregnancy; vitamin B_{12} has a special relationship to the nervous system. Vitamin B_{12} itself was isolated during a search of liver extracts for the material that produces a remission in pernicious anemia. There is a plentiful and cheap supply, and the material has become available for general use.

Megaloblastic Anemia: In the treatment of the average case of megaloblastic anemia of sprue, of infancy or of pregnancy, vitamin B_{12} is not as efficient as folic acid. In fact vitamin B_{12} does not induce a hemopoietic response in the usual case of macrocytic anemia of pregnancy or of infancy. It is clear that many patients need both folic acid and vitamin B_{12}. Folic acid is composed of pteroic acid and glutamic acid, and, when these materials are administered separately or together, the body cannot fabricate them to make folic acid, thus showing that folic acid is a true vitamin and must be prefabricated outside of the body. When given in massive doses, thymine (5-methyluracil) produces a hemopoietic and clinical response similar to folic acid. The folic acid content of the following foods is high: fresh, dark green, leafy vegetables; liver; cauliflower; kidney; beef; veal; and dry wheat breakfast cereals. The following foods are low in folic acid content: root vegetables; tomatoes; bananas; milk; cheese; pork; ham; lamb; rice, corn meal; and sweet potatoes.

Arthritis: In the four years that have elapsed since corticotropin and cortisone were first used as therapeutic agents, so much has been written on them that it often must be difficult for the practicing physician to evaluate their usefulness in the day-to-day practice of medicine. In order to do this, he first must realize that the adrenal cortex is vital to daily living in protecting the human being against many changes in his environment. Although the proper use of these hormones requires a great deal of scientific information, it is desirable that the practicing physician use them. By judicious use, they have been found to be invaluable therapeutic agents, as is illustrated in the following case history:

Case 5.—A 40-year-old coal-loading machine operator was admitted to the clinic complaining of arthritis of nine years' duration. Until nine years previously he considered his health to have been unusually good; then he twisted his left shoulder while operating a coal-loading machine. A roentgenogram of this shoulder revealed nothing abnormal at that time. His shoulder was taped, and he was given heat therapy, but the pain was so severe that he was given sedatives to relieve it. After five or six days the pain subsided sufficiently to enable him to return to work, but it was not relieved completely. About a week later intense pain suddenly developed in the right groin, and he was admitted to a hospital and given injections to relieve the pain. After nine days in the hospital he was discharged. Since then, the pain had "jumped" from one joint to another and by this time had involved almost every joint in his body. Every two or three weeks he had an acute attack of pain, and, when his joints became swollen, red, and hot, the pain was so intense that he was unable to work for four or five days. Five years prior to his admission he gave up working in the mines, and since then he had done odd jobs when he felt able to work. During the nine years of his illness he had had many kinds of therapy. Hot mineral baths had given him more relief than anything else he had tried.

Physical examination showed a well-developed, well-nourished man with good color. His finger joints were slightly enlarged and tender. There was slight atrophy of the intrinsic muscles of the dorsum of the hands. The elbows were slightly enlarged. The right shoulder was tender and slightly swollen. There was diffuse swelling and slight tenderness of the right ankle, and there was tenderness through the distal part of the right foot. The left ankle was slightly swollen but not tender. Over and just below the left elbow were two groups of multiple nodules; each group was about 1¼ in. in diameter. There was a large firm nodule on the left heel and a firm nodule on the bottom of the left foot. There was a smaller

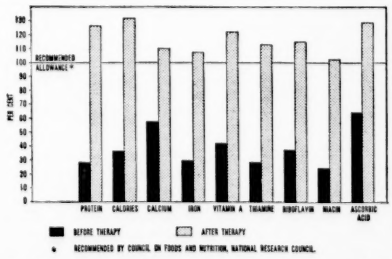

Fig. 9.—Nutrients supplied by diet of patient with rheumatoid arthritis before and after cortisone therapy.

nodule on the right elbow and a similar one on the left wrist. The sedimentation rate was 50 mm. per hour, corrected. Roentgenograms showed decalcification of the bones and swelling of soft tissues around the affected joints. All other laboratory findings were within normal limits.

He was given a total of 1,900 mg. of cortisone acetate (Merck) intramuscularly in a period of six days. The pain in the involved joints began to subside three hours after the first injection; his hands began to "relax," and there no longer was any tension in his shoulders and elbows. He slept more that

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night than he had in a long time. By the following morning the swelling in his right ankle had subsided and he could close his hands completely for the first time in several years. He stated that he never had had as much relief from any other therapy. Gradually all the swelling and soreness of the joints disappeared, so that he could walk and move around with ease, and the nodules became softer. His sedimentation rate decreased to 30 mm. per hour with correction.

For the past four years he has been given therapy intermittently either with cortisone acetate (Merck) intramuscularly or orally or with corticotropin (Armour) intramuscularly. In from four to five days to a month some pain and discomfort return, but they never have been so severe as before the first period of therapy, and he is able to work regularly driving a coal-loading truck. He eats a great deal after therapy with either corticotropin or cortisone acetate, as shown in figure 9. Without treatment his sedimentation varies between 40 and 50 mm. per hour, corrected, and after therapy it decreases to about 17 mm. per hour, corrected. This patient, like many others in the clinic, cannot work without treatment. When enough therapy is given, the symptoms disappear; his strength and appetite return, with the result that he is able to do heavy manual work and support himself and his family.

When one looks at the major causes of death in 1900 and contrasts them with the major causes of death in 1950 in the light of the studies that I have just reported, it is obvious that heart disease, carcinomas, and vascular lesions of the nervous and renal tissues should be approached from the nutritional and metabolic point of view. We propose to do this at our institute in Chicago.

SUMMARY AND CONCLUSIONS

In our studies in the past 25 years, which are touched on briefly here, we have stressed that the lack of proper growth and development of infants and children is a chemical and clinical problem and that the process of aging and illness should be approached as a chemical as well as a clinical problem. Deficiency diseases in human beings tend to develop slowly, and what many physicians fail to realize is that they are corrected slowly. All too frequently the patient and the physician become impatient because they do not realize the time necessary to restore physiological function that was damaged by a slow and long-drawn-out process. Frequently there is a point beyond which there is some irreversible change.

More should be known of the chemistry of disease. When it is, physicians will not be content to accept the traditional view that when a person is not well, or as one ages, he must expect to lose strength, keenness of vision, and alertness and to look and act "old." The physician can never get away from the challenge to think. There will be no automatic or mechanical diagnosis in the near future. The physician who has an uncanny sensitiveness associated with a capacity for memory and experience is needed more in this field than in any other. He must have in mind that there is nothing constant in the human body. The nutrients ebb and flow into the cells. They all bear potentials for change. In this ever-shifting, ever-renewing of the constituents of the body, the cells must be renewed or there will be deterioration. These forces shape the patient's destiny.

As an outgrowth of 25 years' work, the following four essentials for therapy in nutritive failure have been devised: (1) diet—adequate calories, adequate protein, vitamins, and minerals in suitable and edible form for the individual person; (2) basic supplement—vitamin A, thiamine, riboflavin, nicotinamide, ascorbic acid, folic acid, vitamin B_{12}, and "activator" when necessary [8]; (3) additional medication—as indicated for coexisting diseases; (4) natural B complex—dried brewers' yeast powder or liver extract.

The basic formula for the treatment of clinical syndromes of mixed vitamin deficiencies is as follows:

	Daily	
Folic acid	5 mg.	
Thiamine	10 mg.	
Nicotinamide	150 mg.	
Riboflavin	10 mg.	
Ascorbic acid	150 mg.	
Vitamin B_{12}	10 to 15 mcg.	(Usually very large amounts are needed unless highly potent activator is used)

Pregnancy, lactation, growth, and aging result in special stresses on nutritional processes of the body. It is well known that improper diet is not the sole cause of dietary deficiency diseases but that impaired absorption from the alimentary tract, failure of utilization by the tissues, and inadequate storage may be contributing factors. I have tried to stress that the problems presented by patients with deficiency diseases vary greatly and that it is necessary for the physician to give careful consideration to the problem presented by each patient and to prescribe the type of therapy best suited for him. The treatment must restore health as quickly as possible and must prevent recurrence of the disease. The physician should prescribe, in addition to specific therapeutic agents, a diet that the patient can eat and digest satisfactorily. Sole reliance on dietary therapy is inadvisable and impractical. It is better to prescribe too much of the therapeutic agents indicated rather than too little, too soon rather than too late. Precise diagnosis allows one to avoid futile treatment.

Use of nutrients to replenish the tissues and to prevent disease is the physician's primary problem. He knows that the nutrients must be supplied for the body from the outside. No statistics can determine the loss or no words can describe the waste that occurs when physicians do not meet this challenge head on. In unpublished observations by Dr. Robert E. Stone and myself, we have shown that synthetic cortisone acetate (Merck), pituitary corticotropin (ACTH) (Armour), and corticotropin zinc phosphate (Organon) each profoundly affects the nutritional processes. Many severely ill patients have been observed to double their food intake soon after taking any one of these substances.

The little that is known and the much that should be known lead to confusing and contradictory information. It should be recalled that the chemical equilibrium of the human body is preserved in many ways and that the maintenance of this equilibrium becomes more and more difficult as the stores of protective substances are depleted by various disease processes. It is clear that many persons have placed too narrow a significance on the importance of nutrition and metabolism in the general physiological process. Patients must have an adquate intake of the needed nutrients, which must be properly assimilated and utilized, if the physician is to be expected to take the best care of them.

620 S. 20th St. (3)

8. "Activator" is intrinsic factor that must be given in adequate amount to form conjugate of vitamin B_{12} present.

PATHOGENESIS OF CONSTRICTIVE PERICARDIUM

Eli Moschcowitz, M.D., New York

We are well conversant with the clinical and anatomic aspects of the fully developed constrictive pericardium, but, so far as I am aware, no study has been made of the pathogenesis or the clinical phases of the early and intermediate stages. Inasmuch as the course of constrictive pericardium covers many years, the opportunity to observe its development from the beginning to the terminal phase must come but rarely. For the most part, we are forced to reconstruct the pathogenesis by a study of the anatomy and incidence of pericardial adhesions under all conditions. Such a program obviously necessitates an inquiry into the still unsettled cause of constrictive pericardium. With these objects in view, I investigated the autopsy findings and some of the clinical aspects of all the cases of adherent pericardium found at post mortem in the Mount Sinai Hospital during the past 25 years.

DEFINITION

A constrictive pericardium is one in which the two layers of the pericardium are universally adherent and thickened in such a way that they interfere with some of the normal functions of the heart, especially diastolic filling. In addition, torsion of the heart clockwise or counterclockwise on its long axis[1] and compression of the entering veins by the adhesions act to produce cardiac insufficiency and/or lessening of the stroke volume.

Although there is no doubt that adhesions between the parietal and visceral layers of the pericardium represent the earliest anatomic phase of constrictive pericardium, a totally adherent pericardium alone does not imperil cardiac function to any appreciable degree. Postmortem records are replete with instances of uncomplicated, complete synechia of both layers even in elderly patients who have never suffered from cardiac distress. This lesion has been termed accretio cordis and, by some observers, has been sharply differentiated from concretio cordis or constrictive pericardium. Such a distinction, I believe, is not justified, and I shall try to show that sometimes accretio cordis is the earlier stage of constrictive pericardium.

Constrictive pericardium is nearly always associated with adhesions from the external surface of the pericardium to the surrounding structures, either the sternum, the adjacent surfaces of the pleura, the diaphragm, or the mediastinum; also, there are more or less generalized pleural adhesions. Frequently the peritoneum is thickened and adherent, especially on the convex surfaces of the liver and spleen. These manifestations can be attributed to a previous polyserositis the causes of which will be discussed later. To what degree the adhesions from the pericardium to the surrounding structure cause interference with function still remains to be determined. Hosler and Williams [2] performed various experiments on dogs; in some, they sewed the parietal pericardium to the diaphragm, and, in others, they sewed the two layers of the pericardium together and then to the diaphragm. In both instances, circulatory disturbances did not ensue, and tolerance to vigorous exercise on a treadmill was normal. The animals were observed over a period of two and a half years. Postmortem examination revealed no cardiac hypertrophy.

Clinically, constrictive pericardium, in its advanced stages, is associated with a rather classic syndrome. The syndrome has been admirably summarized by Burwell and Blalock.[3] 1. The venous pressure is high and persistent. 2. The systolic pressure is low; the pulse pressure is low; and there is pulsus paradoxus. 3. The heart rate is rapid and becomes excessive on exercise. 4. Both fluoroscopy and kymography show a diminution of excursion of the ventricle that is more marked in the right side. 5. The total blood volume is increased. 6. The velocity of blood flow (arm to tongue and arm to carotid) is prolonged. 7. The output of the heart per minute is diminished. To these we may add anasarca, especially ascites. This syndrome is not pathognomonic for constrictive pericardium; it also occurs in cases of tricuspid stenosis and prolonged cardiac tamponade. But it must be emphasized that it only occurs in cases of full-blown constrictive pericardium and, therefore, cannot serve as a diagnostic approach to the early stages. Aside from the pericardial lesion itself, investigations on the finer morbid anatomy of the visceral lesions have been desultory. Emphasis has hitherto been almost entirely centered on the clinical and operative aspects of the disease.

The persistent, high venous pressure so characteristic of constrictive pericardium would lead one to infer that phlebosclerosis must result not only in the systemic but also in the portal veins. Such indeed is the case. Phlebosclerosis of the hepatic veins, which I described many years ago in association with hypertension of the pulmonary circulation,[4] was found in every one of our patients with constrictive pericardium. In a study of congestive splenomegaly,[5] I tried to show that the essential lesions, namely, fibrosis of the pulp (Billroth) cords, sinus hyperplasia, and fibroadenia, occurred only in persistent hypertension of the portal circulation, whether the result of cirrhosis of the liver, obstruction of the portal or splenic veins, or any cardiac disorder in which a persistent venous pressure is demonstrable. The fibrosis of the pulp cords was interpreted as a capillary sclerosis. These morbid changes were also present in every case of advanced constrictive pericardium in this series. In a recent study of cardiac fibrosis (cirrhosis) of the liver,[6] I tried to show that the fibrosis is largely the result of the simultaneous association of prolonged hypertension of both the pulmonary circulation and the portal circulation. As a result, phlebosclerosis of the hepatic veins is engendered with capillary sclerosis of its tributaries, in the outflow tract of the organ, and phlebosclerosis and capillary sclerosis of the tributaries of the portal vein, in the inflow tract. This lesion also occurred in all our cases

From the Laboratories, Department of Pathology, the Mount Sinai Hospital.

Because of space limitations the bibliographic references have been omitted from THE JOURNAL and will appear in the author's reprints.

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of constrictive pericardium. Finally, I have shown [7] that arteriosclerosis of the pulmonary artery and sclerosis of the alveolar capillaries arise only after there has been a persistent hypertension of the pulmonary circulation, as in mitral disease, emphysema, prolonged congestive failure, pleural adhesions, obliterating lesions of the lungs (tuberculosis and silicosis), and communications between the right and left sides of the heart. In consequence of the prolonged, elevated venous pressure, hypertension of the pulmonary circulation must arise in constrictive pericardium. This has been demonstrated by cardiac catheterization.[8] Every case of constrictive pericardium in this series has shown sclerosis of the pulmonary capillaries manifested by thickening and occasionally by hyaline degeneration, with secondary dilatation.

The association of visceral capillary sclerosis with either phlebosclerosis or arteriosclerosis in organs in which afferent or efferent vessels are subjected to increased arterial pressure (lungs, pancreas, and spleen) or to an increased venous pressure (liver) is a universal principle [9] and is the cause of the increased fibrosis in these organs under such conditions. These changes are viewed as compensatory to the increased pressure. These capillary scleroses of the lung, the liver, and the spleen, with their attendant scleroses of the main afferent and/or efferent vessels, are, other factors being excluded, the distinctive features of constrictive pericardium and serve as anatomic criteria to differentiate accretio cordis from constrictive pericardium. The constriction may be just enough to induce capillary sclerosis, and, if the venous hypertension is not excessive and, especially, if it is not too prolonged, the patient may be entirely asymptomatic. The constrictive pericardium thus becomes an anatomic but not necessarily a clinical entity.

This differentiation in terms of morbid anatomy is emphasized because constrictive pericardium is currently viewed as a combination of both anatomic and clinical characters, and its diagnosis is not considered valid unless both are present.

ETIOLOGY

Inasmuch as the cause of constrictive pericardium is closely allied to that of adherent pericardium, I investigated the cause of all our cases of adherent pericardium. Only the cases which the adhesion was dense and scarlike were included; those in which it was fresh or intermediate were omitted. In 9,618 autopsies, such adherent pericardiums occurred 130 times, or in 1.4%.

On review of these 130 cases, it was found that the causes of adherent pericardium, complete or partial, were rheumatic fever, 46.2%; coronary thrombosis, 18.5%; tuberculosis, 3.1%; no apparent cause (as determined by history and autopsy), 25.5%; polyserositis with indeterminate endocarditis, 4.5%; polyserositis without endocarditis (associated with a typical scleroderma), 0.7%; and syphilis, 1.5%. Of the syphilitic patients, the Wassermann reaction of one was 4+, and the other had a typical hepar lobatum. The pericardial lesions were not structurally syphilitic. In the same 130 cases, the pericardium was completely adherent in 71 (54.6%). The causes, in this group included rheumatic fever, (38), 53.5%; coronary thrombosis (6), 8.5%; tuberculosis (3), 4.2%; no apparent cause (19), 26.8%; polyserositis with indeterminate endocarditis (4), 5.6%; and miscellaneous (1), 1.4%. If rheumatic fever is considered to be a cause of a polyserositis, the latter caused complete obliteration of the pericardial sac in 64.4% of all cases.

The term polyserositis requires a stricter definition, because it has been applied indiscriminately to include both exudates and transudates. I have limited the term to include only those that are strictly inflammatory. Of the six cases, five were classified as indeterminate or atypical endocarditis as described by Libman and Sacks.[10] In one, polyserositis was associated with a scleroderma but not with endocarditis. It is worthy of note that the pericardium, pleurae, and peritoneum were involved in six. If we include the cases of tuberculous pericarditis and rheumatic fever, which strictly speaking may be classified as polyserositis since all three serous cavities were affected, we note that polyserositis, exclusive of instances in which its origin is unknown, accounts for a completely adherent pericardium in 64.4%. I think that polyserositis of unknown origin associated with the type of endocarditis described by Libman and Sacks is important because it may be one of the causes of a constrictive pericardium, an observation that has not been made before. In one of my cases, clinical and anatomic constrictive pericardium was previously classified as of unknown origin. Postmortem observations on cases of endocarditis described by Libman and Sacks, whether associated with lupus erythematosus or not, reveal an unusually intense polyserositis characterized by an unusual quantity of gelatinous mucoid exudate in the pericardium and pleurae and on the surface of the liver and spleen. It is very possible that healing of such a polyserositis may result in development of the anatomic characteristics of constrictive pericardium. Obviously, only a postmortem examination can disclose this variety of polyserositis, and, therefore, no etiological classification of constrictive pericardium can be completely valid without a study of the morbid anatomy of the cardiac valves.

Of the 130 cases of adherent pericardium, a constrictive pericardium in an anatomic but not necessarily in a clinical sense occurred in 7, or 5.4%. Only four conformed to the classic clinical and anatomic criteria of the disease; that is, they represented the most advanced stages of constrictive pericardium. The remaining three cases may be viewed as examples of the early phases; first, because anatomically they possessed many characteristics of constrictive pericardium and, second, because they showed the characteristic capillary sclerosis in the lungs, liver, and spleen described previously.

REPORT OF CASES

Case 1.—A 49-year-old woman gave a history of ascites, dyspnea, and anasarca that had been present for three years. She had had many paracenteses. Physical examination showed marked ascites and venous pressure of 16.5 cm. H_2O that rose to 22 mm. on right upper quadrant pressure. The circulation time was 18 seconds and the ether time 8 seconds. Pericardiectomy was performed, with perforation of the right ventricle. Hemorrhage occurred, and death followed.

Autopsy showed polyserositis; total adhesive pericarditis, with calcification; both pleurae adherent and thickened; chronic perihepatitis; chronic perisplenitis; thickening of parietal peritoneum; ascites; phlebosclerosis of the vena cava and hepatic veins; cardiac fibrosis of the liver; chronic passive congestion of the spleen, with splenomegaly; bronchopneumonia; and varicose veins of the mesentery of the small intestine. The

heart weighed 425 gm.; the right auricle was slightly dilated; and the left ventricle was contracted. The pericardium was thick and calcified throughout. The spleen weighed 40 gm. The liver weighed 1,490 gm. and was fibrotic.

Microscopic examination revealed that the alveolar capillaries of the lungs were thickened and dilated (capillary sclerosis); many heart failure cells. The pericardium was very thick, hyalinized, and calcified. The liver showed advanced cardiac fibrosis, and the intima of the hepatic vein and vena cava was greatly thickened. The spleen showed a typical early phase of congestive splenomegaly, namely, sinus hyperplasia, fibrosis of pulp cords, fibroadenia; and a thickened and hyalinized capsule.

This patient presented the typical clinical and anatomic manifestations of a constrictive pericardium. The history and autopsy, however, revealed no obvious cause.

CASE 2.—At the age of 24, this man had severe upper quadrant pain with fever. On exploratory laparotomy, the gallbladder was found to be normal. Eight months later, he was reexplored because of severe pain in the right lower quadrant. Only a much thickened capsule of the liver (Zuckerguss) and some abdominal fluid were found. At the age of 39, the man was hospitalized because of ascites and bilateral pleural effusions. The venous pressure was 25 cm. H_2O. Pericardiectomy was performed, but the patient died two days later.

The findings at autopsy included constrictive pericardium; perforation of the right ventricle; pleural adhesions and thickening; arteriosclerosis of pulmonary artery; cardiac fibrosis and chronic congestion of the liver; marked hypertrophy and dilatation of both auricles; hypertrophy of both ventricles; intimal thickening of hepatic veins and superior and inferior vena cava; anasarca; hydrothorax; indeterminate endocarditis of aortic valve; and bilateral healed apical tuberculosis. The pericardium was dense, firm, and 1 cm. thick. The right auricle was greatly dilated and hypertrophied; the tricuspid valve was dilated. On the edge and anterior surface of the right posterior aortic cusp there was a gray-pink, wart-like nodule that measured 3 by 2 mm.

Microscopic examination of the lungs showed capillary sclerosis with many heart failure cells. The pericardium was greatly thickened and hyalinized. The wart-like nodule at the aortic valve was densely fibrous. The liver showed centrolobular necrosis, and the intima of the hepatic veins and inferior vena cava was markedly thickened. The spleen showed sinus hyperplasia, fibrosis of the pulp cords, and lymphadenia. The capsule was thickened. This patient presented a typical clinical syndrome of constrictive pericardium confirmed both by operation and autopsy, with typical pulmonary capillary sclerosis, cardiac fibrosis of the liver, phlebosclerosis of hepatic veins and inferior vena cava, and congestive splenomegaly. The indeterminate endocarditis described by Libman and Sacks leads to the suspicion that there had been an asymptomatic nonspecific polyserositis in the past, an association that is quite common.

CASE 3.—This 25-year-old woman had had "heart trouble" since youth, with numerous episodes of congestive failure. A roentgenogram of the heart showed extensive calcification of the pericardium. Pericardiectomy was performed twice, four and two years earlier, with some relief after each operation. Eventually the patient died in congestive failure.

Autopsy, after resection of the sternum and pericardiectomy, revealed rheumatic heart disease; chronic adhesive calcified pericardium; chronic valvulitis of the mitral valve, with stenosis and insufficiency; chronic valvulitis of the aortic and tricuspid valves; dilatation and hypertrophy of both auricles and ventricles; bilateral pleural adhesions; chronic, passive congestion of the viscera; bilateral pleural effusion; ascites; chronic perihepatitis and perisplenitis; arteriosclerosis of the pulmonary artery; and cardiac fibrosis of the liver. The heart was much enlarged. The pericardium averaged 1 cm. in thickness and was calcified throughout. The pericardium was adherent to the anterior chest wall, adjacent pleurae, and mediastinum. The valves showed lesions typical of rheumatic fever, but no Aschoff's nodes were found. The alveolar walls of the lungs showed capillary sclerosis; the alveoli contained heart failure cells. The liver showed severe cardiac fibrosis. The spleen showed fibrosis of the pulp cords and some sinus hyperplasia. There was marked perisplenitis and perihepatitis.

This woman presented the clinical manifestations of constrictive pericardium and multiple valvulitis of rheumatic origin. Microscopic examination showed capillary sclerosis of the lungs, the liver (cardiac fibrosis), and the spleen (congestive splenomegaly). To what extent these lesions were the result of the constructive pericardium or of the associated valvular disease, it is impossible to say. In all probability, one was superimposed on the other as suggested by the severity of the microscopic lesions.

CASE 4.—The patient was a physician in whom pulmonary tuberculosis and a pericardial effusion developed eight months before his hospitalization. The liver and spleen were enlarged. The venous pressure was 23 cm. H_2O. Pericardiotomy and pericardiectomy were performed during which an enormously thick pericardium was removed. Biopsy from the anterior surface of the pericardium demonstrated tuberculosis. After the operation, the venous pressure dropped to 5.5 cm. H_2O; later, an empyema was drained. Continuous fever and cachexia developed, and the patient died.

Autopsy, seven and one-half months after partial pericardiectomy and pericardiotomy, revealed chronic tuberculous pericarditis; miliary tuberculosis of the lungs, kidneys, and spleen; extensive bilateral pleural adhesions; hypertrophy of the left side of the heart and arteriosclerosis of the aorta and systemic arteries (the protocol did not mention whether the pulmonary artery was arteriosclerotic); granular perihepatitis; hydronephrosis and caseous tuberculosis of left kidney; tuberculosis of mediastinal lymph nodes; and splenomegaly. The heart was enlarged, and there was marked hypertrophy of the right side of the heart and of the left ventricle. The pericardial sac was distended. The wall was between 5 and 7 mm. thick. The external surface was adherent to the adjacent pleura. The capillaries of the pulmonary alveolar walls were dilated and thickened, and the alveoli contained heart failure cells. The surface of the liver was covered with fibrous deposits and showed central congestion. The portal spaces were filled with inflammatory cells. The spleen showed some sinus hyperplasia and slight fibrosis of the pulp cords. All the organs showed many tubercles.

As in case 3, the clinical phenomena were so dominated by tuberculosis that any symptoms referable to constrictive pericardium were largely suppressed; nevertheless, capillary sclerosis in the lung and in the spleen indicated that constriction of the pericardium was active.

CASE 5.—As a child, this 14-year-old boy had measles and frequent colds. He had always been underdeveloped and had had diarrhea since the age of seven. The stools were foamy and bulky, and the boy had secondary anemia. He was admitted to the hospital one year before the present admission. At that time, his condition improved on a diet high in carbohydrate and low in fat content supplemented with iron and liver extract. His liver then was large, and his spleen was palpable two fingerbreadths below the free border of the ribs. His present admission followed an appendectomy performed five days previously. He died with symptoms of intestinal obstruction.

Autopsy showed acute ileus caused by an omental band; chronic ulcerative colitis; parietal thrombi in the superior mesenteric and portal veins; chronic interstitial hepatitis; splenomegaly (the spleen weighed 420 gm.); and chronic adhesive pericarditis, with phlebosclerosis of the inferior vena cava and hepatic veins. The heart was normal in size, and the valves were intact. The pericardium was 0.5 cm. thick, and the pericardial cavity was completely obliterated. The lungs showed dilatation and thickening of the alveolar capillaries and a few heart failure cells in the alveoli. The liver showed phlebosclerosis of the hepatic veins and inferior vena cava and slight interstitial hepatitis. The spleen showed fibrosis of the pulp cords, sinus hyperplasia, and marked congestion. There were numerous fibrous thrombi in the submucosal vessels of the large intestine with beginning organization and large parietal thrombi in the superior mesenteric and portal veins.

The pericardial lesion was surely independent of the ulcerative colitis. While symptomless, the thickened and totally ad-

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herent pericardium must have caused hypertension of the pulmonary circulation as manifested by the pulmonary capillary sclerosis and the heart failure cells. Also there must have been persistent peripheral venous pressure as evidenced by phlebosclerosis of the hepatic veins and of the inferior vena cava and hypertension of the portal circulation as manifested by an early, congestive splenomegaly. The slight interstitial hepatitis was probably of nutritional origin. Had the boy lived, the probability is strong that the clinical evidences of an advanced constrictive pericardium would eventually have developed. No cause for the totally adherent pericardium was discovered.

CASE 6.—A 51-year-old man had pain in the chest with dyspnea, orthopnea, and cyanosis, four weeks before admission. The blood pressure was 124/70 mm. Hg. The liver extended five fingerbreadths below the free border of the ribs. The venous pressure was 18 cm. H_2O. The electrocardiogram resembled that of a coronary closure. The patient was treated with mercurial diuretics but gave no response.

At autopsy the pericardium was completely obliterated and partially calcified. There was primary carcinoma of the liver; carcinomatous invasion of the right hepatic and the portal vein; massive occlusion of the right pulmonary artery; multiple emboli in the lungs; dilatation of the right auricle and ventricle; cardiac fibrosis of the liver; multiple pleural adhesions; and metastatic nodules in both lungs. The pericardium was greatly thickened and hyalinized and contained a calcific girdle 0.5 cm. thick that enveloped the middle and much of the ventricular portion of the heart. Except for a note of the presence of emboli, the pulmonary arteries were not described in the protocol.

On microscopic examination, the pulmonary capillaries were dilated and tortuous; the liver showed cardiac fibrosis and phlebosclerosis of the hepatic veins; and the spleen showed congestion but no congestive splenomegaly.

This case may be interpreted as one of anatomic constrictive pericardium of unknown origin, and, although symptomless, it was sufficient to cause hypertension of the pulmonary circulation as manifested by capillary sclerosis of the lungs and persistent systemic venous hypertension as evidenced by cardiac fibrosis of the liver and phlebosclerosis of the hepatic veins. Had death caused by carcinoma not intervened, I believe that the clinical manifestations of a constrictive pericardium would have developed. No cause for the pericardial lesion was discovered.

CASE 7.—A 17-year-old boy had had weakness for three months, cough for three weeks, and fever and signs of fluid in both sides of the chest and abdomen. The spleen was palpable. Twelve weeks before his admission, his family physician discovered signs suggestive of pericarditis. He had dyspnea on exertion, orthopnea, and edema of the lower extremities. Congestive failure developed, and the patient died.

Autopsy revealed bilateral pleural effusion; ascites; complete obliteration of pericardial sac; tuberculous pericarditis; disseminated tuberculosis of the lungs, spleen, kidneys, and lymph nodes; chronic, passive congestion of the liver and phlebosclerosis of the superior and inferior vena cava and of the hepatic veins; arteriosclerosis of the pulmonary artery; mediastinal adhesions; and peritoneal tuberculosis. The pericardium averaged 1.5 cm. in thickness. It was hyalinized, and, on its inner aspect, there were many caseating tubercles.

Microscopically, aside from tuberculosis, the lungs showed sclerosis of the alveolar capillaries; the liver showed central congestion; and there was phlebosclerosis of the hepatic veins and of the superior and inferior vena cava. The spleen showed sinus hyperplasia and fibrosis of the pulp cords; in other words, congestive splenomegaly.

I believe this case demonstrates anatomically an early, constrictive pericardium. The presence of pulmonary arteriosclerosis and alveolar capillary sclerosis shows that there was prolonged hypertension of the pulmonary circuit. Phlebosclerosis of the hepatic veins and of the inferior vena cava and the early phase of congestive splenomegaly all indicated that there was sustained peripheral and portal venous pressure. The clinical phenomena were so masked by tuberculosis that it is impossible to say to what extent the constrictive pericardium was responsible for the symptoms.

COMMENT

An analysis of these seven cases provokes a number of interesting deductions. First, it is obvious that constrictive pericardium, in the anatomic sense, does not always give rise to the classic clinical phenomena. Inasmuch as the clinical evidences are mostly the results of the morbid visceral anatomy as revealed in the vascular and capillary scleroses, especially of the lung, the liver, and the spleen, induced by prolonged venous hypertension both systemic and portal, one may conclude that the intensity of the clinical phenomena is largely dependent on the duration of venous hypertension. The degree of venous hypertension is probably of less importance. I have shown in studies on congestive splenomegaly [5] and cardiac fibrosis of the liver [6] that these lesions are dependent more on the duration than on the severity of venous hypertension. Second, in regard to etiology, my observations are not entirely in accord with those of other observers. In three cases, no cause was obtained from the history or at autopsy. One was associated with rheumatic fever, and, while there is no absolute proof that the constrictive pericardium was a result of this disorder, the probability is strong that it was the result of a rheumatic polyserositis. Two cases were undoubtedly caused by tuberculosis, and, in one, the presence of an indeterminate endocarditis of the type described by Libman and Sacks suggests that a nonspecific polyserositis preceded the constrictive pericardium.

Paul, Castleman, and White [11] described 53 cases. In 56.5%, no cause was discovered. Tuberculosis was demonstrated in 17%. There was no sharp distinction between the microscopic picture of healed tuberculous pericarditis and so-called idiopathic constrictive pericardium. In two cases, healing tubercles were found during surgery, but no evidence of tuberculosis was found at post mortem two and three years later. In none of the 53 cases was there a history of rheumatic fever. In three cases there was a history suggestive of pericarditis some years previously.

Martensen and Warburg [12] report rheumatic fever in 2 out of 25 cases. Tuberculosis was verified by histological examination in three cases; in two other cases, it was probable. In two, constrictive pericardium was due to trauma; in one, to blunt violence and, in the second, to a gunshot wound. In the remaining cases, the cause was obscure. In this connection, Strauss [13] reports a case in which constrictive pericardium was due to a needle left in the pericardium for seven years after diagnostic aspiration. Joest [14] states that foreign bodies have been ingested by cows and, after ulcerating through the diaphragm, have come to rest in the pericardial sac and produce a septic inflammation with total synechiae, hyalinization, and calcification resulting in a constrictive pericardium. Blalock and Burwell [15] found the tubercle bacillus in 21 of 28 cases in which the cause could be established by operation, aspiration, or autopsy. Staphylococci were found in 3 of the 28. In an earlier publication,[3] these same authors found not a single instance caused by rheumatic fever. McKissick [16] reports 20 cases of which he regarded 8 to be caused by tuberculosis as evidenced by a history of prolonged exposure to tuberculosis, by biopsy of the pericardial scar, or by the presence of tuberculosis in other sites, such as the bones, lymph nodes,

or lungs. In the other 12, the cause was unknown. McKissick also reports verification by biopsy of a tuberculous pericardial effusion that, after 20 years, developed into a constrictive pericardium. Of 24 cases, Harrington [17] found tuberculosis confirmed by biopsy in 5. In the remaining 19, the cause was unknown. Sellars [18] found tuberculosis in 16 out of 20 cases of constrictive pericardium. In a later publication,[19] he described 18 cases of tuberculous pericarditis. In 4 of these, no other serous cavity was affected, and, in 16, constrictive pericardium developed.

Apparently there is a wide discrepancy in these reports with respect to the incidence of the various causes. There is no doubt that tuberculosis is a fairly common cause and that probably over half the cases reveal no ascribable cause. There is a very wide divergence with respect to the incidence of rheumatic fever as a cause. In one of our seven cases, rheumatic valvulitis was found at autopsy. The reason our findings differ from those of other workers with respect to etiology is perhaps that we have investigated only postmortem material. Our study does not answer the question of why certain cases of polyserositis result in constrictive pericardium and why others do not. The probability is strong that the result depends on a number of factors; the intensity of primary polyserositis, its duration, and the reactive potentialities of the organism.

SUMMARY AND CONCLUSIONS

The pathogenesis of seven cases of constrictive pericardium, which came to postmortem examination, is described. In four of these (cases 1 through 4), the classic clinical signs and symptoms of constrictive pericardium were apparent. Most of the clinical phenomena may be attributed to sustained hypertension of both the pulmonary circulation and the peripheral venous system, including the portal circulation. As a result of sustained hypertension, there is arteriosclerosis of the pulmonary artery, sclerosis of the vena cava and hepatic veins, and sclerosis of the tributary capillaries of the lungs, liver, and spleen, with the development of sclerosis of the pulmonary capillaries, cardiac fibrosis of the liver, and an early phase of congestive splenomegaly. Three patients (cases 5 through 7) lacked many of the signs and symptoms of contrictive pericardium but had many of the anatomic characters, namely, an unusually thick, totally adherent pericardium. In one case, the pericardium showed considerable calcification and, in a second, complete hyalinization. In addition, there were extensive adhesions to the surrounding pleurae, arteriosclerosis of the pulmonary artery, perihepatitis and perisplenitis, and phlebosclerosis of the hepatic veins and vena cava. There was sclerosis of the pulmonary capillaries in two cases, cardiac fibrosis in one, and an early phase of congestive splenomegaly in two. Three cases may be viewed as disclosing potential constrictive pericardium; in other words, as an earlier phase. Etiologically constrictive pericardium is one of the late results of polyserositis whether from tuberculosis or rheumatic fever or associated with an indeterminate endocarditis, possibly of the type described by Libman and Sacks. Only a postmortem examination is decisive in determining the precise cause.

25 W. 68th St. (23).

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PRIVATE PRACTICE IN SMALL INDUSTRY

Forrest E. Rieke, M.D., Portland, Ore.

For too many years an ignored paradox in American industry has encouraged an unfortunate parallel in American medicine. In this age of huge corporate manufacturing organizations, only recently has the vast size and great importance of so-called small industries been recognized. In government and business much has been said about the necessity for aiding and preserving the "little fellow," but, because of scattered location and a genius for individual differences, the "little fellow" has not submitted well to organization for aid.

In the field of industrial medicine it has been possible to develop highly coordinated and skilfully staffed medical, surgical, and health conservation programs for big industrial groupings. Similar services have not been readily assembled for small industry.[1] Brief reference to figures shows both the urgency and size of the "small plant problem." National statistics have been drawn together by Klem and McKiever [2]; from this source the table in the next column is taken. The table shows that 99% of employee organizations have less than 500 persons, and 70% of total employees are found in so-called small plants. With minor variations these percentages may be applied to most states and large and small cities. In Portland, Ore., in 1947,[3] with a population of 350,000, 80% of the workers were employed in units of less than 500 persons and 55% in units smaller than 100; these figures are substantially the same today.

Size of Class (No. of Employees)	Reporting Units		Workers	
	No. (in Thousands)	Percentage	No. (in Thousands)	Percentage
All units	2,734.2	100.00	35,805	100.00
1 to 49	2,637.9	96.48	14,219	39.71
50 to 499	88.9	3.25	11,085	30.96
500 to 999	4.4	0.16	3,012	8.41
1,000 and over	3.0	0.11	7,489	20.91

WHAT IS THE SMALL INDUSTRY MEDICAL PROBLEM?

Accident rates are more than twice as high in small plants, and sickness disability rates are relatively unimproved in such units. Medical consultation is not accessible and infrequently used by the small plant in the fields of placement examination, job transfer, rehabilitation, psychological evaluation, case finding, and utilization of the aging worker. These and other random

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1. McCahan, J. F.: Medicine + Industry = Occupational Health, New England J. Med. **247**: 470-472 (Sept. 25) 1952.

2. Klem, M. C., and McKiever, M. F.: Small Plant Health and Medical Programs, Bulletin 215, United States Public Health Service, 1952.

3. Rieke, F. E.: Evolution of Small Plant Health Service as a Private Practice, Indust. Med. **19**: 301-312 (July) 1950.

statements are frequently applied, and all are readily substantiated.

Definition of the problem is complicated by absence of a suitable description of the small plant. For my purpose it can be described as the employee group, too small to have afforded or provided physician or nurse service, predominantly composed of less than 250 persons. It is more apt to be a group numbering 20 to 100 employees.

In many places and publications one can find excellent descriptions of industrial medical and health services. When one attempts to apply these services, prohibitive difficulties have been encountered. Most troublesome features of the small plant health problem are to be found in these areas: (1) unawareness that private physicians, primarily in general practice, are in fact the only existing industrial service for most small plants, (2) unfamiliarity of private medical practitioners with industrial health needs and methods, (3) disinterest in improvement of preventive and therapeutic services for industry by these same private practitioners, (4) unfamiliarity of small plant owners with such services and ineptitude at searching for same, (5) unwillingness of owners and many insurance carriers to spend to prevent and conserve in lieu of expenditure to correct illness and injury, (6) failure of community medical agencies and services to foster and coordinate physician-employer ventures in this realm, and (7) a lingering attitude among physicians that service in industry is not as noble, worthy, or ethical as other forms of medical services.

There is much hopeful development as well as discouraging apathy in all of these zones. In an attempt to illuminate problems, methods, and goals and encourage others in private practice to extend efforts to small plants, the following plan is recorded from work in small industries at Portland, Ore. An attempt will be made to relate our experience to the difficulties mentioned.

GENERAL PLAN

In previous articles [4] wartime and postwar transition in Portland's industries was described with its shift of manpower from six huge shipyards to thousands of smaller employee groupings. After the war, in an effort to continue "large industry" medical services akin to those in the war industries, two industrial zone dispensaries were opened in the center of shop areas. These have been operated as a private venture, staffed with physicians and graduate nurses, and maintained solely to provide the services needed by small plants in the surrounding territory. They have not ventured into other medical fields.

Major emphasis has centered on: (1) emergency treatment of industrial injury, (2) diagnosis of minor and incipient ailments and wide referral to private physicians of those with nonoccupational illness, (3) placement and other special examinations relating to job problems and toxicology, (4) collection of industrial medical and toxicological data as reference material for plants, physicians, and nurses, (5) rehabilitation for injured and ill, (6) case finding through examination and survey, (7) plant sanitation, industrial hygiene control, and accident prevention, and (8) consultation with any group seeking help in the improvement of employee health and safety.

In diagnosis and treatment of more extensive industrial injury and occupational illness, full use has been made of a local general hospital and consulting services of all available medical specialties. A high degree of cooperation has been forthcoming from public and private health agencies, most insurance carriers, and most employers. Employee response has varied from apathetic to enthusiastic; repeating business has been gratifyingly frequent.

With the exception of those sent by the employer for placement examination, the employee has not been obliged to come to the dispensaries for services. Absence of compulsion or contractual obligation in either direction has been most worthwhile, obviating abuses ofttimes arising in contract practice. Fees for service are levied against insurance carriers, employers, or the patient, depending on nature of the ailment. Lack of a prearranged retainer has, of course, been a barrier to full use of consultation by small operators.

PROBLEM AREAS

Unfamiliarity of Profession.—As inferred, the majority, if not all, of the industrial medical service reaching employees in small plants comes from physicians devoting most of their time to general practice or the specialties, and certainly not to the specialty of industrial medicine. In the smaller communities physicians often know the details of local industry. In medium-sized and larger cities this is not usually the case. Employers and workers repeatedly complain that physicians in these localities show little awareness of job requirements, injuring forces, chemistry of the job, the impact of illness on the worker, and the dislocation of production caused by his absence. In the dispensaries in Portland there has been some difficulty utilizing physicians as currently prepared for any but the most limited work in relating medical and health factors to demands of the job. Preparation in surgical trauma is good, but work in rehabilitation and conditioning of patients for return to work are unfamiliar to many. It seems that medical schools have not yet managed to acquaint those entering practice with the needs and methods of industrial medicine, a rather glaring deficiency that most schools are doing little to overcome. There is also an evident need for extensive effort to acquaint those already in practice with the same subjects.

Disinterest of Practitioners.—Lack of interest of practitioners in an improved industrial service stems from both physical separation in point of geography and in a fundamental preoccupation with the individual person removed from his environment. Rarely do medical buildings adjoin the industrial work place, and rarer still do physicians visit the work place. Most physicians are greatly stimulated by such visits, and service to the employee-patient is materially improved when his physician knows the daily environment as well as specific chemicals, processes, and hazards in this environment. The employer and employee are pleased when a physician pays a "house call" to his patient's work place. Furthermore, the employer is a willing ally in the ferreting out of noxious occupational agents and assisting in

4. Rieke, F. E.: Health Service for Very Small Plants Offered Through Private Practice, Occup. Health **12**: 127-129 (Aug.) 1952. Rieke.[4]

recuperation and rehabilitation when the informed physician phones or writes, seeking help for his patient. Even the unglamorous physical examination gains interest when the physician knows that the worker may be exposed to certain extremely toxic chemicals, or required to make hundreds of exact spatial movements per hour or to drive a 30 ton truck on narrow roads in reduced illumination for six or more hours nightly. The influence of aggravating noise, constant danger, dulling monotony of work, or galling supervision on the psychosomatic disease can be weighed only by the physician interested enough to know of their existence in the patient's daily life.

Unfamiliarity of Small Plant Owners.—With rare exceptions small plant operators have had no experience with industrial service. When the service exists their desire to purchase it must be considered very low. After six years in the field, there are only small beginnings in use of preventive services other than those in the early care of trauma. The beginnings exist, however. Through observation of our work for larger employee groups, through education by employer organizations, by the chamber of commerce, and by the state board of health, the "little fellows" are becoming aware of their needs and are seeking examination and consultative services. The medical society's role in this regard will be mentioned later.

Failure to Pay for Values Received.—The small plant operator rarely has a budget, to say nothing of planned financial provision, for medical services. Most are unfamiliar even with the cost of accidents as represented by premiums to insurance carriers. Most insurance carriers have not successfully educated the smaller plants in the need for expenditure on preventive medical services. Thus far, so-called union health plans have been equally unproductive in this direction in the Portland area. Insistence by many insurance carriers in industry that their insured merit a reduction in fees by treating physicians has gone far to dampen medical enthusiasm for industrial service. Absence in local offices of insurance carriers of medical consultants and the routine processing of bills and case reports by clerical personnel utterly unfamiliar with medical methods and problems further alienate the interested physician. A further insistence by employers on a very low fee for examination of the employee imparts the conviction to both physician and employee that a competent and searching examination is not desired. On the other hand, the recent trend for employers to ask and pay for a very thorough examination, the earnest effort of many insurance carriers to secure fullest treatment and rehabilitation of the injured, and efforts to arrive at mutually agreeable fees by conference with medical societies all warm the atmosphere in which medical service is delivered and provide a healthier culture medium for growth of interest and fuller physician service. In general, physicians believe that industry deserves the best service available, delivered at fees on a par with those charged other responsible persons and groups in the community. When industrial services are justly valued accelerated growth can be expected. Parenthetically, a most useful summary has been prepared recently by Dr. R. B. O'Connor [5] listing types, costs, and results to be expected in small plant health service.

Failure of Community Group Leadership.—Until recently too few medical societies have exerted leadership or encouraged extension of preventive services in industrial areas. At times there has been actual criticism and obstruction of those toiling in this field. Under the spur of the Council on Industrial Health of the American Medical Association this attitude is changing. Local and state societies have established industrial health committees, and community health councils number many representatives from management and labor.

In Portland a joint committee of the state medical society and the chamber of commerce has held one larger dinner meeting for employers and insurance, medical, nursing, and safety workers, and planning is underway for an area-wide annual conference on industrial health and safety. It is hoped that this may emulate the successful Houston, Texas, conference and other similar efforts. The Oregon state medical society has appointed a liaison committee to meet with representatives of the insurance industry, and it is hoped that this may prove to be an avenue for mutual instruction in the duties and functions of physicians and insurance carriers in their relation to all insured. One of the honored 1952 Sommer Memorial Lectureship speakers brought semiyearly to Portland to address the medical profession was Dr. Rutherford Johnstone, who spoke to large audiences on occupational disease in its various relationships. He stirred a genuine interest among specialists and general practitioners alike.

Recent conferences between the American Medical Association Industrial Council and the Academy of General Practice have clearly illuminated the large potentials for service by this group in industry. A plan is under consideration to include considerable industrial information in postgraduate activities of the academy, and this emphasis will certainly expedite wider participation by its membership.

ETHICAL PRACTICE IN INDUSTRY

Ethics of American medicine exist and are formulated to guarantee a continuing high level of honest, selfless, and devoted service to the public. As times change, methods of practice necessarily change. It is well to remember, therefore, that what is new or strange is not necessarily unethical. As medicine has built on its foundations in the basic sciences, it has added to the bacterial concept of disease and aseptic techniques of surgery the sure knowledge that physical and mental health are best sustained by prevention bulwarked by ever more effective therapeutics. For preventive medicine to be effective it must reach well persons as certainly as the ill. To do this the practitioner must often be the aggressor, and aggressiveness in medicine is frequently considered unethical and may be deleterious. It has been felt, however, that the private practitioner who is critical of those who carry the preventive fight against disease and injury into the limelight of industry is guilty of sins of omission in his too great concentration on treatment of disease. It is hoped that he too will go on the offensive in psychosomatics, case finding, health education, and leadership for health.

5. O'Connor, R. B.: Small Plant Health Programs—Types, Costs and Results, Occup. Health **12**: 123 (Aug.) 1952.

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Contacts with industry are an ethical extension and improvement of office practice. There is just criticism of those who use industry only as a builder and feeder of practice. Uncondoned also is the pirating of selected patients through industrial contacts. Part of industry's resentment toward medicine arises because many physicians have built a practice in industry and then withdrawn further service. It is to be hoped that private practitioners will increase rather than diminish efforts through industry.

Contacts with employee groups are legitimately made in many ways. In Portland the dispensary's services are introduced through explanatory talks at business and service clubs, patients served in the dispensary and hospital, and other physicians, health agencies, and insurance carriers. Requests are welcomed for new or different services whether for flight or truck operators, lead workers, or meat processors. The work place is visited, and, if puzzled about diagnosis or unsatisfied about work situations, the physicians go to the shop on their own initiative. This is well received and is considered good ethics, for in no other way can the patient's problem be solved. Under patterns described, wherein the medical society and industry are joining hands to become acquainted, worry over legitimacy of industrial service and ethical contacts will be greatly ameliorated.

COMMENT

There are those today who have great plans for the private practice of medicine, whether it be to make it "free," to finance it by prepayment only, to furnish it by groups only, or whatever. I am not among these; I subscribe to the theme so well phrased by Alexander Woollcott when he said, "For the architects of any utopia are inconvenienced by discovery (usually at the last minute) that the human race is not, as at present matured, equipped for the exacting role forced upon it by citizenship in Utopia."

I am impressed by the truly remarkable viability and flexibility of American free enterprise as exemplified medically in the private practitioner. His system is indigenous and for the most part well adapted to local needs. Changing needs have been well met before, and it is reasonable to assume that as methods are improved and needs clarified the small plant industrial problem will be well solved by the practicing American physician.

SUMMARY AND CONCLUSIONS

A private practice serving the medical needs of many small plants in an American city of 350,000 is described. It is observed that the small plant problem can be met best by enlargement of the scope of private practice to fill the preventive and treatment needs of employers and employees. This will require an interested curiosity by practitioners and improved cooperation with public and private health agencies. Expansion of educational activities is urgently needed by medical schools and physician associations to familiarize physicians with industry's needs and methods to fill them. Similarly, wide increase of joint medical society–industry efforts will aid small plant ownership to utilize the private practitioner more effectively. A more honest financial return to physicians in the service of industry will go far to win friends and create a climate for growth. Medical society sponsorship of service for industry will remove many doubts about the ethics involved.

210 A. N. E. Oregon St.

SURGICAL SEPARATION IN CRANIOPAGUS

Herbert J. Grossman, M.D., Oscar Sugar, Ph.D., M.D., Paul W. Greeley, M.D.
and
Max S. Sadove, M.D., Chicago

On Sept. 16, 1951, craniopagus twins were born in Rock Island, Ill. The record of the birth and course during the first six weeks has been adequately described by Durr.[1] The present preliminary communication will give a résumé of the sequence of events that led to the eventual surgical separation of the twins and of the postoperative course.

The babies were admitted to the University of Illinois Hospitals on Oct. 2, 1951. They were in a conjoined state of craniopagus parietalis (fig. 1). Their general condition was good, with normal increments of growth and neuromuscular development. Their measurements on the Stuart-Vickers tables ranged from the 25th to the 50th percentile for their age. The babies had obviously independent activities; they slept at different times, could eat at separate times, made separate movements, had independent bowel and bladder functions, and had individual body temperatures. In all, there was clinical evidence of two autonomous nervous systems. There was a mirror-imaged moderate asymmetry of the head (including the face), the right side of R. L. B. and the left side of R. D. B. being more prominent.

Both babies had systolic murmurs, which were loudest to the left of the sternum at the level of the third interspace. It was considerably louder in intensity in R. D. B. There was no evidence of impaired cardiac function. The left side of the chest of R. D. B. was larger—corresponding to the asymmetry of the face. This was mirrored in R. L. B. The heart sizes were normal. The murmurs were believed to represent an intraventricular septal defect.

The problem at hand was whether or not a surgical separation could be attempted. Unfortunately there was little material in the literature that was of any pertinent

The drawings and photographs were prepared in the Illustration Studios of the University of Illinois.

From the departments of pediatrics, neurology and neurological surgery, and surgery, divisions of plastic surgery and anesthesiology, University of Illinois College of Medicine.

1. Durr, S. P.: Craniopagus Twins, J. A. M. A. **150**:93 (Sept. 13) 1952.

value. The few previous attempts at separation of other craniopagus twins had failed.

Before an actual separation could be attempted, the following basic questions had to be answered: 1. Could the babies withstand numerous diagnostic and operative

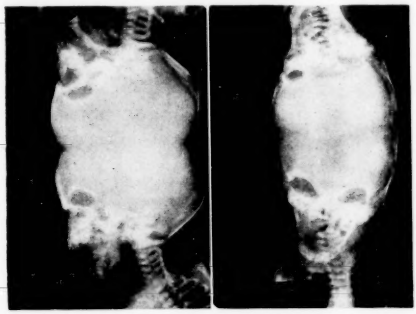

Fig. 1.—Roentgenograms of the skull made at the age of 5 days. Note absence of bony septum and over-riding of parietal bones in the antero-posterior view.

procedures? 2. Were the brains separate? 3. Was there a dural septum? 4. Were the cerebral arteries separate? 5. Were the venous sinuses separate? 6. Could the anticipated defects in the scalp and skull be closed? Except for the obvious malformation of a conjoined state, results of all studies, including roentgen examination of the chests and long bones, blood studies, and urinalyses, were within normal limits. The systolic murmurs of neither child appeared to be of any functional significance; their general physical condition was good.

Clinical observation of independent activity indicated the probability that the brains were functioning inde-

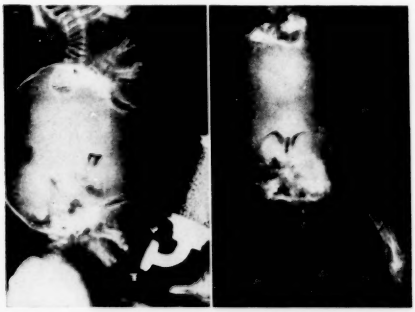

Fig. 2.—Pneumoencephalograms made at the age of 6 weeks. Air was injected into lumbar subarachnoid space of R.D.B. The subarachnoid air is confined to one brain. Note the height of the third ventricle between lateral ventricles. (There was questionable agenesis of the corpus callosum.)

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Carotid angiography was carried out after operative exposure of the common carotid artery (the right carotid artery of R. D. B. and the left carotid artery of R. L. B.). Direct intra-arterial injection of iodopyracet (Diodrast) permitted visualization of the intracranial circulation of R. D. B.; owing to technical difficulties, adequate films were not obtained for R. L. B. The films show (fig. 3) independent arterial circulation: the delayed films showed dye in the venous return of only the baby in whom injection had been made. There was no way of accurately predicting from these films what the venous

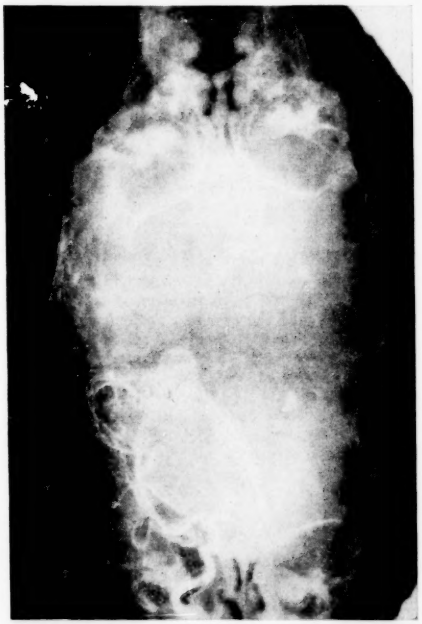

Fig. 3.—Angiogram in R.D.B. at age 5 months. Iodopyracet (Diodrast) in the middle cerebral artery distribution appears to cross over to the opposite hemisphere, but the midline dye is actually from the anterior cerebral arteries. No dye appears in the cerebrum of R.L.B.

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PREPARATION OF SKIN FLAPS

If separation were accomplished, adequate covering for the exposed intracranial structures would have to be provided. It is well known that any free skin graft will

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grow on exposed brain or dura. It will adhere tightly, however, and such a covering would not be satisfactory, for it would later become necessary to insert some structural support between the skin covering and dura in order to protect the brain. Transplanted skin and subcutaneous fat, but not a free graft, could be reelevated in the future (much as one might do with a normal covering), the mechanical support inserted, and the soft tissue covering then replaced. Since skin with its underlying fat will not grow as free graft, some type of pedicle flap arrangement had to be planned in order to maintain its own viability.

Although specific information concerning the venous drainage systems was inadequate, it was agreed that separation should be attempted. The skin flaps were prepared first. Many different places were considered as the source of the flaps. The most logical solution seemed to be to use direct circumferential flaps taken from around the head. They could be transferred very simply, without discomfort and without the necessity of immobilization in awkward positions. The base of the flaps, in the occipital area, could be brought to the forward part of the skull opening and thus provide some hair where it was most needed. The donor areas from which the flaps were taken could be covered readily with free skin grafts, with which an acceptable cosmetic result might be anticipated.

When the use of scalp flaps had been decided on, it was found by actual measurement that each flap would be approximately 14 in. (35 cm.) long by 3 in. (7.5 cm.) in width. This is very long as compared with the width, especially when the normal blood supply coming in from the vertical axis would be cut off and thus make maintenance of nutrition of these narrow flaps still more hazardous. All of this could be done successfully, however, if sufficient patience and time were used. Repeated "delaying" operations would give adequate circulation if enough time elapsed between the various stages. Each stage would cut off more and more blood supply coming in through the main arteries, namely the frontalis, supraorbital, and superficial temporal arteries of the two sides. As this source of nourishment was gradually diminished, it would simultaneously stimulated increased vascularity rising from the base of each flap in the occipital area and also produce a new additional blood supply in the transverse axis, i. e., parallel to the linear direction of the flap.

The foregoing plan was started on May 14, 1952 (fig. 6, part 1). After three stages, the scalp flaps were completely divided around their peripheries. During the next five months (occupied by several neurosurgical diagnostic procedures) large transverse veins appeared in each flap. These proved to be of inestimable value in preventing venous congestion at the time of transfer. During the first attempt at separation on Nov. 26, 1952, the scalp flaps were elevated completely from their beds. Their blood supply was excellent even though both were 14 in. (35 cm.) long by barely 3 in. (7.5 cm.) wide.

Very early in the course of our observations of the babies, it was evident that there would be numerous diagnostic and surgical procedures, some of which would require an anesthetic agent. During all procedures there was an anesthesiologist with a separate set of instruments (anesthesia machines, laryngoscopes, endotracheal tubes, etc.) with each infant. In each anesthetic procedure, the infants were given only enough of some basal anesthetic agent to produce a state of sleep. This was supplemented by inhalation anesthesia when indicated. Local anesthesia was used as the primary technique where applicable utilizing basal anesthesia by the rectal route with tribromoethanol (Avertin), thiopental (Pentothal), or paraldehyde, and the inhalation of a mixture of nitrous oxide, oxygen, and trichloroethylene as a supplement where necessary. In this way psychic trauma was avoided as the children fell asleep and knew nothing about the procedure. Intubation with soft rubber endotracheal tubes was used in all procedures in which manipulation would endanger the airway.

The respiration of each infant was supplemented or controlled as individually required. At all times, the aim was to have the minimal toxicity at the time of operation and minimal residual toxicity, in order to diminish the possibility of damage from repeated anesthesia. During the operations, continuous consultation between the anesthesiologists and the pediatricians permitted proper fluid replacement. Even in operations done with a small amount of local anesthesia, there was integration of management by all concerned.

In the major procedures that required long periods of time, the problem was magnified by the blood loss and the danger of trauma to the airway from having the tube in place for as long as 12½ hours. The danger of trauma was accentuated by the manipulations necessary during surgery. The problem was one of maintaining the vital functions throughout. The anesthesia was maintained at a light plane to avoid cumulative damage, but yet had to be deep enough to prevent interference from movement of the patients. The chief agent used was nitrous oxide with as high an oxygen concentration possible. Trichloroethylene was added only for a few minutes at a time to reestablish adequate depth of anesthesia.

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For the first attempt at separation, an incision was made at the junction of the frontal and parietal bones (right side of R. D. B., left side of R. L. B.), and a channel 1.5 cm. wide from the vertex to the occiput was made. An attempt was then made to split the dura mater into its two fused portions. This proved to be impossible beyond a depth of about 1 cm., because the two leaves of dura mater were fused into one sheet; furthermore, as dissection continued in this plane, there was considerable venous bleeding, the source of which could not be determined. Accordingly, incisions were made on either side of the midline septum, exposing the subarachnoid space on either side of the septum. It could be seen that the dural septum did, in fact, separate two brains; however, because of the peculiar position of the two heads and their immobility with respect to each other, it was impossible to look into the depths on either side of the septum. The dural septum was then incised toward the midline between the two brains. This allowed visualization, at a distance of some 6 cm., of two longitudinal sagittal sinuses that did not appear to communicate with one another in the frontoparietal region, which was the limit of the exposure at this time. This exploration and the antecedent elevation of the scalp flaps had taken six

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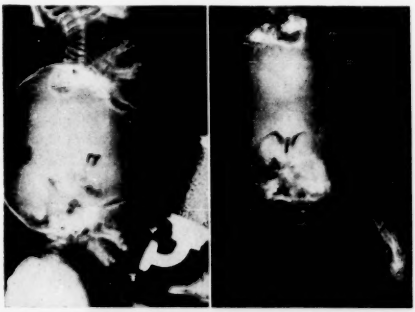

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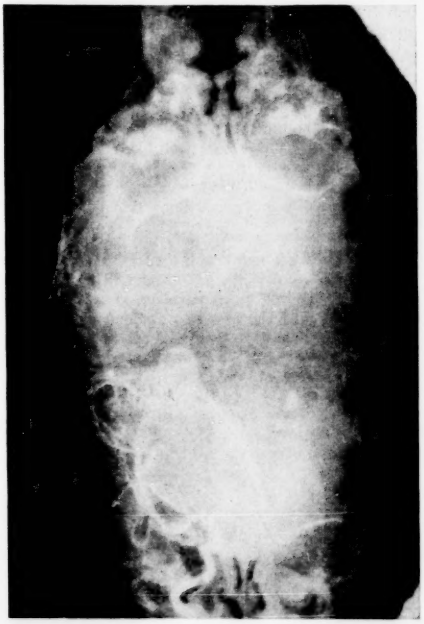

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and a quarter hours, and it was felt that complete separation could not be accomplished then and that the procedure should be terminated. The incised dura mater was sutured at its periphery, and the wounds closed. The scalp flaps were resutured into their original beds. The postoperative convalescence was uneventful, except for a short left focal seizure in R. D. B., involving the arm, and lasting less than a minute, that was followed by a mild transient hemiparesis for about 18 hours.

FINAL SEPARATION

The final separation was made on Dec. 17, 1952. This was started at the frontal junction with an incision made parallel to the dural septum and a few millimeters from it, to avoid the bleeding encountered when separation of the dura mater had been attempted in the earlier operation. It was believed that the dural defect created by going on R. L. B.'s side of the septum might be covered with polyethylene film. The falx of R. L. B. was found and cut while the numerous vessels were clipped as necessary. After a few centimeters of exposure, it became evident that, in order to permit visualization into the depths, it would be necessary to clip the veins running from the surface of the brain to the dural septum. This would inevitably interfere with the venous return from one of the twins. By the time this information was obtained, there was enough interference with the brain of R. L. B. that it was clear that complete separation would not markedly increase the impairment of cerebral function already anticipated (fig. 4). Accordingly, the falx was cut completely, and the veins that drained the surface of R. L. B.'s cerebrum were also clipped and cut as they entered the dural sinuses. These were found to

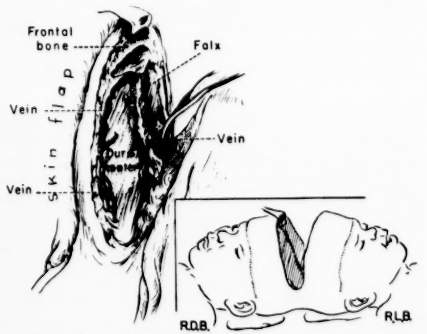

Fig. 4.—Artist's drawing of findings during final separation (age 15 months).

be three in number (fig. 5); they lay within the dural septum. One was far out in the periphery, forming a semicircle on the left side of R. D. B.'s brain, and there was one on either side of the cut falx, which had extended from R. L. B.'s brain to the dural septum. It was possible to reach the posterior part of the falx where there were huge venous lakes connecting all of the lateral sinuses and all of the sagittal sinuses, as well as the circumferential sinus posteriorly in what amounted to a conjoined torcular Herophili. With the incision being made on R. D. B.'s side, these enormous conjoined sinuses were cut through. Hemostasis was obtained with difficulty, and the babies went into shock, which was counteracted by liberal administration of blood.

Thus was accomplished the complete separation of the two infants, 10½ hours after the operation was begun. R. L. B.'s brain had the typical appearance of congestion with petechial hemorrhage that would be expected from a brain with an intact arterial supply but virtually no venous outflow. R. D. B.'s brain appeared to

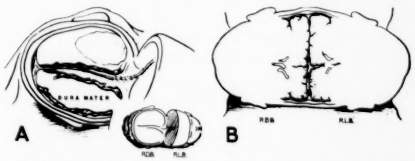

Fig. 5.—*A*, diagram of arrangement of abnormal dural venous sinuses as disclosed by operation. Note the dural defect over the right hemisphere of R.D.B. *B*, reconstruction of arrangement of dural venous sinuses, seen in coronal section. Note the apparent absence of the falx in R.D.B.

be normal except for some congestion and edema in the right parietal region where the dural septum was defective, presumably as a result of failure of healing of the incision made in the dural septum on that side in the first separation attempt.

The problem now arose as to how to cover the denuded brains, which had no dura mater. Polyethylene film was used for this purpose; it was sutured to the dural edge of R. L. B.'s brain and to the dural and/or subcutaneous tissues over R. D. B.'s brain. The blood loss had been so great and the operation had taken such a long time that it was felt unwise to consider reflecting the scalp flaps and placing them in position on top of the polyethylene film, for this would have meant more hours of operating time and more loss of blood that might not have been withstood. It had originally been planned to put polyethylene plates in place at this time, but the molding of these plates and their anchoring into the wound would again have been too onerous a burden, so this, too, was omitted and we were content to use sterile aluminum foil to cover the top of each head before putting on dry dressings and sterile bandages. The aluminum foil was easily molded, could be sewed, and was believed to be mildly bacteriostatic. The entire procedure required 12½ hours. R. D. B. received 2,500 cc. of blood and R. L. B. 3,500 cc. during procedure.

After separation, R. D. B. cried spontaneously; R. L. B. showed attempts at crying while the endotracheal tube was still in place. A small catheter placed in the tube revealed an obstruction. The quick removal of the tube and tracheobronchial aspiration showed that the accumulation secretions had become rubber-like, blocking the airway. After this resuscitative procedure, the baby cried weakly.

Course of R. D. B.—At the time of separation when it was necessary to separate the venous plexus, there was a sudden loss of blood that resulted in a severe cardiovascular collapse in R. L. B., so that no pulse, blood pressure, or heart sounds were detectable. The very rapid administration of oxygen by pressure on the breathing

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bag, positive pressure on the thorax, and rapid intravenous infusion of blood restored essentially normal cardiovascular function. He did not respond to painful stimuli. About eight hours after operation, there was marked irregularity of rate and depth of respiration, with retraction of the suprasternal and infrasternal regions. Secretions from the hypopharynx were aspirated easily, with the patient in an atmosphere rich in oxygen and moisture, but signs of right upper pulmonary atelectasis appeared. Although it was considered probable that much of the respiratory difficulty might be due to central nervous system derangement, tracheotomy was done by Dr. Cecil Riggs of the department of otolaryngology, to provide as open an airway as possible. Periodic breathing of the Biot type appeared, however, indicating a central nervous system cause for the respiratory difficulty. Accordingly, the head dressings were removed, when it was seen that the swelling and engorgement of the cerebrum seen during the operation had progressed. Relief of pressure by removing the artificial coverings of the brain brought immediate improvement in breathing. The tachycardia observed during the Biot breathing became slower.

With the long exposure of the brain, contamination and possible infection were anticipated, so penicillin and streptomycin were given intramuscularly. Organisms sensitive to these antibiotic agents were suppressed, but an infection with Pseudomonas aeruginosa was detected on the fourth postoperative day. This was controlled by daily local applications of 2½% polymyxin B to the surface of the brain and by intramuscular injections of 2 mg. per kilogram of body weight per day. No evidence of systemic toxicity was discovered, although the antibiotic was given until his death 34 days after separation. By this time, the surface infection of the brain was completely controlled. Staphylococcic organisms were controlled by intermittent administration of aureomycin, oxytetracycline (Terramycin), and erythromycin. Eleven days after operation, signs of diffuse bronchopneumonia appeared, with marked expiratory difficulty and very tenacious secretions. In order to liquefy these, water was nebulized over the tracheotomy tube. Since high humidity alone proved insufficient, a solution of trypsin (Tryptar) was given as an aerosol through the nebulizer (20,000 to 50,000 units of trypsin in 2 to 3 cc. of diluent at ph 7.4 every six to eight hours. On several occasions, Alevaire (a solution of glycerine, sodium bicarbonate, and a detergent, Triton WR-1339, in distilled water) was used in a similar manner. Marked liquefaction of the secretions resulted, permitting their aspiration and resulting in marked improvement in respiration. No evident signs of toxicity to the drug were noted. The signs of bronchopneumonia subsided. During the survival period, the infant remained comatose and exhibited signs of decorticate rigidity. Parenteral fluid therapy was given for the first few postoperative days, and thereafter nutrition was maintained by feedings through a no. 19 polyethylene nasogastric tube. Up to 4,500 calories daily were necessary. During the last week of life, there were marked variations in rectal temperature (108 F to 94 F), the peaks not responding to antipyretic measures. Respiratory irregularities of central nervous system origin appeared again on Jan. 19, 1953, and he died on Jan. 20, 1953.

Course of R. D. B.—Immediately after the separation, R. D. B. cried and moved all extremities. He took fluids by mouth the following day and responded to familiar persons. He was kept in oxygen with supersaturated humidity to minimize the effects of the prolonged period of intubation. Supplementary parenteral fluids and antibiotics were given as with R. L. B. On the fourth postoperative day, he was irritable and refused oral feedings. There was an odor from the head dressing characteristic of a Ps. aeruginosa infection, so the dressings were removed, and the surface infection which was found was treated with polymixin B in the same manner and dose as in R. L. B. The thick tenacious exudate typical of infection with this organism gradually subsided. It had not completely disappeared by Dec. 30, when the clinical status of the patients was improved sufficiently to permit proceeding with the plastic surgery (fig. 6). On this day, the scalp flap (fig. 6, part 1) was reelevated and transferred to cover the posterior half of the exposed intracranial structures (fig. 6, parts 2 through 5). The terminal end of the flap was turned to cover part of the anterior defect. All of this flap grew well except for a short segment along the distal edge. This ultimately was trimmed off. After the flap was transferred, the donor site from which it was taken was covered with a large autogenous split-skin graft taken from his back (fig. 6, parts 6 through 8).

On Jan. 17, 1953, the base of the pedicle in the occipital area was divided so as to stimulate additional return circulation from the opposite end of the transplanted flap (fig. 6, part 9). On Feb. 10, the proximal end of the flap was completely detached from the occipital area and transferred forward to cover nearly all of the remaining open defect (fig. 6, parts 10 through 12). Donor sites were covered in each case by split-thickness grafts, some of which had to be revised later.

On March 11, the "dog ear" in the flap was divided in its central portion. The anterior part of the flap was reelevated until it could be shifted anteriorly without tension to cover the balance of the open cranium. The excess "dog ear" was trimmed off and its edges sutured. By this time, the infection was not apparent. This twin, too, showed no signs of toxicity from the antibiotics.

Several days after the separation, when there was more spontaneous activity, it became apparent that there was a left hemiparesis and left astereognosis. Although he was able to hold objects when he looked at them, he did not know what to do with them if his eyes were turned away, nor did he place his extremities properly if he did not see them. The hemiparesis disappeared within the first month, but the astereognosis still continues, although less marked than before. There are no hyperactive stretch reflexes on the left, nor is there any loss of common sensibility. There are intermittent Babinski signs on both sides, which are considered to be normal for his age.

Before the separation, the neuromuscular development of the twins was considered normal. This was assessed with difficulty because of the peculiar nature of the junction of the heads, which undoubtedly interfered with the development of head control, tonic neck reflexes, and the ability to sit and stand. Both had normal infant jargon. During the three months that followed separation, R. D. B. showed only a slow progression of

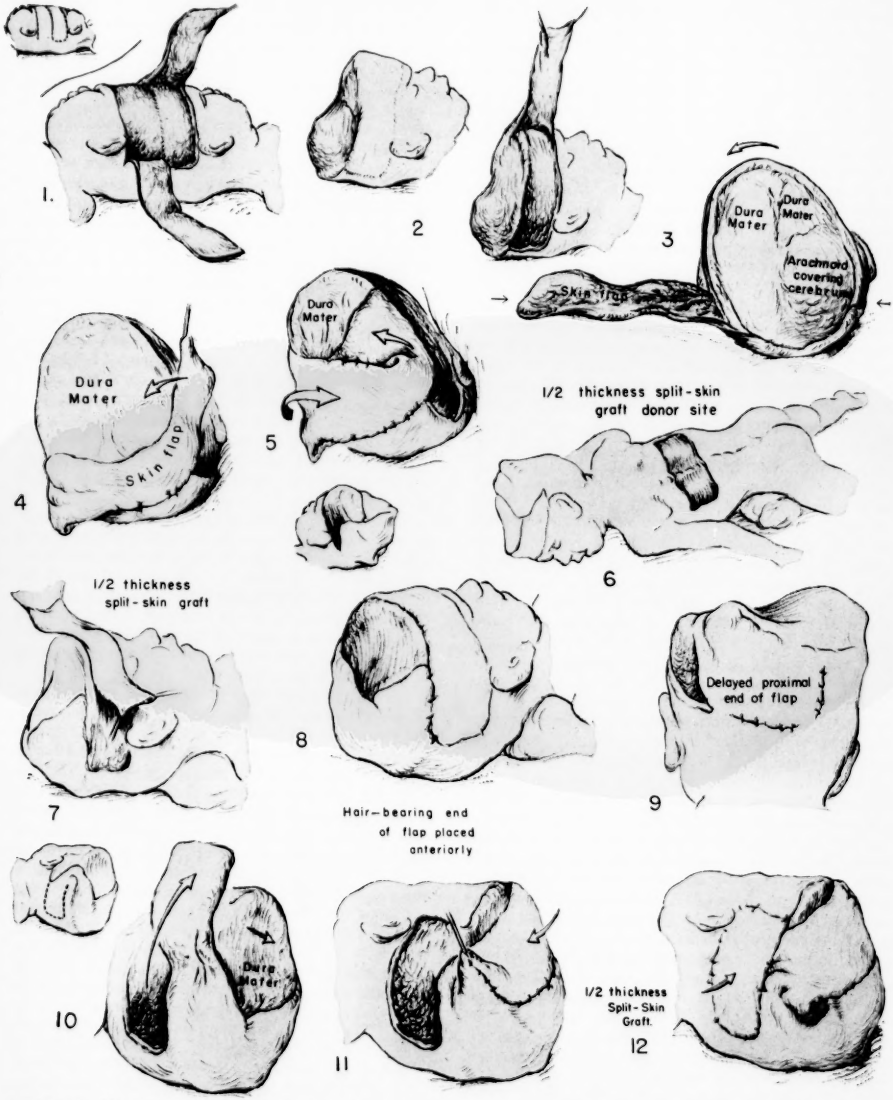

Fig. 6.—Origin of circumferential pedicle flaps of both infants and stages in covering exposed intracranial contents by skin flaps in R.D.B. *Part 1*, pedicle flaps, which were replaced after the first attempt at separation on Nov. 26, 1952. *Parts 2 through 5*, reelevation of skin flaps and transfer to cover the posterior half of the exposed intracranial structures of R.D.B. (Dec. 30, 1952). The arrows in *part 3* indicate the severance site of the terminal end of the flap. *Parts 6 through 8*, covering of exposed donor site of pedicle flap with split-thickness graft from the back of the pedicle flap (Jan. 6, 1953). *Part 9*, extension of the proximal end of the full-thickness flap by delay of circulation to the base (Jan. 17, 1953). *Parts 10 through 12*, completion of the rotation of the full-thickness graft to cover the remaining exposed intracranial structures (Feb. 10, 1953). The donor site is covered by a split-thickness graft (March 1, 1953).

development. This is attributed to the trauma of separation, debility, infection, and repeated operations that took place during this period. Since the head covering has been completed, he has shown a considerable advance in development and improvement in his general physical state. He has had a considerable amount of occupational and physical therapy, which has contributed to this improvement. He can now sit alone, but cannot pull himself into the sitting position. He has good control of his head and good coordination of all extremities, and his jargon has increased in amount and in intelligibility. He is alert, responds to favored personnel, and makes attempts to feed himself. The hip flexors (which were tight, presumably owing to lack of standing) have become normal with physical therapy. It is difficult to assess the degree of development in all spheres because of the many factors, biological, traumatic, and psychological, that have affected his present state.

There is still no solid protection for the brain, and the solution of this problem is now under active consideration. We are reluctant to use any foreign material over such a large surface of dura mater and under skin flaps, because of the hazards accompanying buried foreign bodies. Autogenous bone grafts appear to offer the best replacement for the calvarium that never developed, but the extensiveness of the defect make accomplishment of closure by this means difficult.

All the professional and technical skills have basically one objective—to permit this baby to grow and develop as a happy, effective human being. His handicap—like other handicaps—need not prevent him from fulfilling this goal. It has been necessary throughout, therefore, to provide for him as well as his family an understanding and comfortable environment. Without such an environment—and without the help of his parents and our help to them in understanding his problems—the boy's development might not have proceeded so well. The future planning must involve most careful attention to his emotional and social development. This will consist of his parents' greater and greater participation in his care as his physical problems permit, continued medical and surgical efforts to correct his physical difficulties with as little pain and discomfort as possible, and long-term planning to assure stimulating educational opportunities that are compatible with his capacities. These provisions, associated with increasing understanding of all handicapped children by the public, will provide the best assurance that this boy will have the opportunities that should come to all children—that of the fulfillment of his greatest potentialities as a human being.

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CHOICE OF A CORONARY VASODILATOR DRUG IN CLINICAL PRACTICE

EVALUATION OF EFFECTS BY ELECTROCARDIOGRAPHIC TESTS

Henry I. Russek, M.D., Karl F. Urbach, M.D., Alexander A. Doerner, M. D.
and
Burton L. Zohman, M.D., Staten Island, N. Y.

Each year new drugs are introduced that are alleged to have coronary vasodilator action. After an initial enthusiastic endorsement, most of these agents fall into disrepute or lose popularity because of the uncertainty arising from conflicting claims. Frequently, the clinical value of a coronary vasodilator drug has been estimated from its pharmacological action in animals or from its performance in uncontrolled studies in human disease; less often it has been appraised from carefully controlled analyses of the subjective sensation of pain in patients with angina pectoris. Some attempts to determine the effect of drugs on the severity and frequency of anginal attacks occurring spontaneously or induced by exercise have successfully removed or minimized the element of bias; but even the best of these methods employing the double blind technique with placebo and test materials have left much to be desired. Such procedures would be acceptable for assaying the vasodilator potency of drugs, if pain represented a definite entity and reflected a fairly constant degree of coronary insufficiency in each patient. Obviously, this is not the case. Pain is of infinite variability and to date has not been accurately quantitated in man either subjectively or objectively. Furthermore, even if such precise measurement were possible, pain could not reliably reflect the status of the coronary circulation or the degree of myocardial ischemia. In our laboratory, for example, patients who show constant electrocardiographic alterations with each performance of a standard exercise test under carefully controlled conditions have experienced no distress on some occasions and appreciable pain on others. Conversely, as Dock [1] has pointed out "a minute ischemic area in the myocardium may cause as severe discomfort as ischemia of a whole ventricle." By such methods of evaluation, drugs that only elevate the threshold for pain might be erroneously labelled as coronary vasodilator agents. A more accurate index of coronary insufficiency than that afforded by the subjective sensation of pain alone is, therefore, needed if rational therapy by means of drugs is to be established in angina pectoris.

With this need in mind, we attempted to record and compare the ability of specific agents to modify the electrocardiographic response to standard exercise (Master

Walter A. Brecher gave technical assistance.

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From the Cardiovascular Research Unit, Department of Medicine, United States Public Health Service Hospital, Staten Island, N. Y. A joint project with the National Heart Institute.

1. Dock, W.: Angina Pectoris: The Neuralgic Features of the Classic Form of Heberden's Disease, M. Clin. North America 33:635 (May) 1949.

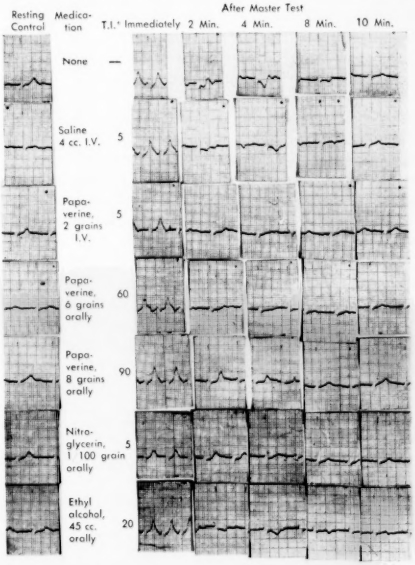
two-step test) in carefully selected persons.[2] To have validity such investigation must include only those patients with coronary disease who exhibit, on repeated testing under identical conditions, a relatively constant positive response to a given amount of exercise. The difficulty in obtaining patients suitable for such study can be estimated from a four year experience in screening over 3,000 patients with coronary artery disease by means of a two-step exercise electrocardiographic test. Of this number, only 52 patients qualified as fulfilling the criteria. In these patients, it was found that appreciable periods exist during which the electrocardiographic alterations evoked by standard exercise recur in almost identical fashion with each performance of the test. Even after weeks or months of observation, it was often necessary only to add or subtract several ascents and descents of the two-step staircase to obtain new controls for further test comparisons. By such adjustments, the inevitable changes attending the natural course of the disease did not hinder continued use of most of these test subjects for bioassay of vasodilator drugs. In this manner, as many as 200 exercise tests were carried out in one patient over a four year period.

MATERIAL AND METHOD

Fifty of the patients in this series were men between the ages of 32 to 77 years. Two were women, 49 and 52 years of age, respectively. Seven of the 52 patients had sustained previous myocardial infarction, and 30 presented classic symptoms of angina pectoris. Arterial hypertension was present in 20 of the 52 subjects. In all patients included in this series, the control response to exercise remained constant when recorded from day to day and, in addition, could be favorably modified by the sublingual administration of therapeutic doses of glyceryl trinitrate (nitroglycerin) just before the test. By establishing this prerequisite, it was felt that a basis for comparison could be obtained between the effect of the potent coronary vasodilator glyceryl trinitrate and that of other agents with unknown or undefined action. Under informal conditions with reassurance, demonstration, and explanation designed to allay the patient's anxiety, these tests were carried out under identical environmental conditions at the same time each day and by the same observers. In most instances, only one test was performed in any 24 hour interval. Each drug was tested three to six times in every patient. The following drugs (see table) were studied: glyceryl trinitrate, papaverine hydrochloride, ethyl alcohol (in the form of scotch or rye whiskey), aminophylline, Roniacol (beta-pyridyl-carbinol tartrate), tolazoline (Priscoline) hydrochloride, tetraethylammonium chloride, octyl nitrite, dioxyline (Paveril) phosphate, visammin (khellin), heparin, bishydroxycoumarin (Dicumarol), pentaerythritol (Peritrate) tetranitrate, and morphine.

Analysis of Results of Various Drugs in Fifty-Two Patients

Drug	Dosage	No. of Tests	No. of Patients	Good Response (Patients)
Glyceryl trinitrate	1/100-1/150 grain	208	52	52
Papaverine	1-2 grains I.V. 3-8 grains orally 1.5 grains orally	38 71 36	10 14 12	7 7 0
Ethyl alcohol	1-2 oz.	62	12	0
Pentaerythritol (Peritrate) tetranitrate	10-20 mg.	64	12	8
Aminophylline	7.5 grains I.V. 6 grains orally	28 18	8 6	1 0
Roniacol (beta-pyridyl-carbinol tartrate)	100 mg. orally	33	8	0
Visammin (khellin)	200 mg. I.M. or orally	41	7	0
Octyl nitrite	1-4 inhalations	92	15	3
Tolazoline (Priscoline) hydrochloride	25-50 mg. I.M. or orally	30	6	0
Tetraethylammonium chloride	300-500 mg. I.M.	24	8	0
Dioxyline (Paveril)	200-500 mg. orally	52	10	2
Heparin	50-100 mg. I.V.	110	18	0
Bishydroxycoumarin (Dicumarol)	200 mg.	21	6	0
Morphine	¼ grain subcutaneously	9	6	0



*Minutes between medication and test

Fig. 1.—Master test responses (lead CF_5) obtained in a patient with angina pectoris with and without the administration of drugs.

RESULTS

Glyceryl Trinitrate.—The 52 patients in this series were tested repeatedly with glyceryl trinitrate, and, on every occasion, a strikingly favorable effect on the response to exercise as recorded electrocardiographically

2. (*a*) Russek, H. I., and others: Influence of Saline, Papaverine, Nitroglycerine and Ethyl Alcohol on the Electrocardiographic Response to Standard Exercise in Coronary Disease, Circulation **1**: 700 (April) 1950. Russek, H. I.; Naegele, C. F., and Regan, F. D.: Alcohol in the Treatment of Angina Pectoris, J. A. M. A. **143**: 355 (May 27) 1950. Russek, H. I.; Regan, F. D., and Naegele, C. F.: 100% Oxygen in the Treatment of Acute Myocardial Infarction and Severe Angina Pectoris, ibid. **144**: 373 (Sept. 30) 1950. Russek, H. I., and others: Effect of Khellin in Coronary Artery Insufficiency as Evaluated by Electrocardiographic Tests, New York J. Med. **52**: 437 (Feb. 15) 1952. (*b*) Russek, H. I.; Urbach, K. F., and Doerner, A. A.: Effect of Heparin in Cases of Coronary Insufficiency: Evaluation by Electrocardiographic Tests, J. A. M. A. **149**: 1008 (July 12) 1952.

was observed (fig. 1 to 3). A normal exercise response was obtained in 39 of the 52 patients when 1/150 to 1/100 grain (0.4 to 0.6 mg.) of glyceryl trinitrate was administered sublingually five minutes prior to commencement of the test. In the remaining 13, although a completely negative record was not obtained after administration of glyceryl trinitrate, significant improvement in the postexercise electrocardiographic patterns was observed in each instance.

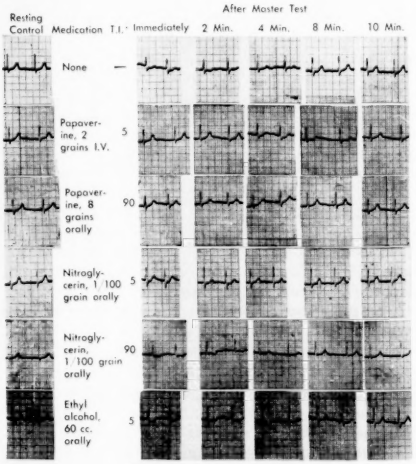


Fig. 2.—Master test responses (lead V_5) in a patient with coronary insufficiency with and without the administration of drugs.

Papaverine Hydrochloride.—Papaverine was administered in doses of 1 to 2 grains (0.06 to 0.12 gm.) intravenously or 1.5 to 8 grains (0.10 to 0.518 gm.) orally to 14 patients in this series. Intravenous administration five minutes before exercise was found effective in 7 out of 10 patients as shown by completely normal responses or improved electrocardiographic patterns as compared to controls (fig. 1 and 2). Large oral doses of papaverine, 3 to 8 grains (0.194 to 0.518 gm.), gave similarly favorable results in 7 of 14 patients who were tested from 60 to 90 minutes after its administration. In contrast, usual therapeutic doses of papaverine showed negligible effect on the electrocardiographic response to standard exercise.

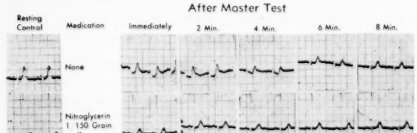


Fig. 3.—Effect of glyceryl trinitrate and pentaerythritol tetranitrate respectively on the response to the Master test (lead V_5) in a patient with coronary insufficiency. Glyceryl trinitrate was given five minutes before the test, pentaerythritol tetranitrate one hour before.

Ethyl Alcohol.—Twelve patients were given ethyl alcohol in the form of scotch or rye whiskey (1 to 2 ounces) 5 to 20 minutes before exercise. Although 62 tests were performed with this drug, not a single instance was noted in which the electrocardiographic response was favorably influenced by its administration (fig. 1 and 2). In striking contrast, it was also found that ethyl alcohol prevented or reduced anginal pain in all seven patients in this group in whom this symptom was evoked during control tests.

Pentaerythritol Tetranitrate.—In a dose of 10 to 20 mg., pentaerythritol tetranitrate was found to exert a marked modifying influence on the electrocardiographic response to standard exercise in 8 of 12 patients tested. In all of the patients showing favorable response, the results were comparable to those obtained with glyceryl trinitrate, but the duration of action was considerably more prolonged. Thus, with pentaerythritol tetranitrate, an improved response could be elicited as long as four to five hours after administration of the drug (fig. 3 and 4).

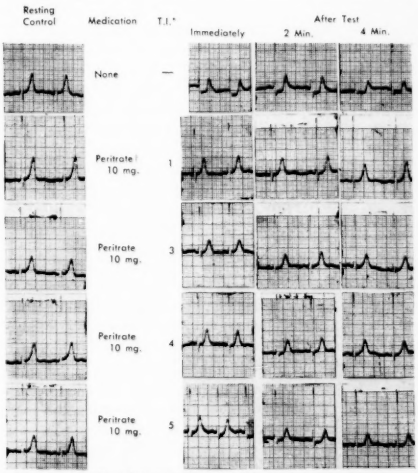


Fig. 4.—Master test responses in a patient with coronary insufficiency after a single dose of 10 mg. of pentaerythritol tetranitrate; normal response obtained as long as four hours after administration of the drug.

Other Drugs.—Aminophylline in the dose of 7½ grains (0.5 gm.) intravenously or 6 grains (0.4 gm.) orally and Roniacol in a dosage of 100 mg. orally were both found to have slight or insignificant effect. Even in massive doses, visammin, 200 mg. administered intramuscularly or orally, failed to alter favorably the electrocardiographic response in all patients tested. Octyl nitrite by inhalation was at best as effective as 1/300 grain

(0.2 mg.) of glyceryl trinitrate. Tolazoline hydrochloride in 25 to 50 mg. doses intramuscularly or orally and tetraethylammonium chloride in 300 to 500 mg. doses intramuscularly showed either no modifying effect or an adverse influence on the response to exercise. Dioxyline phosphate in amounts similar to those employed with papaverine did not give comparable results. Dioxyline phosphate appeared to be a much less potent drug than papaverine according to this method of testing. Heparin in recommended doses (50 to 100 mg. administered intravenously once or twice a week) did not exert a significant effect on the electrocardiographic response to standard exercise in any of the patients tested. Bishydroxycoumarin in therapeutic dosage was also without demonstrable value. Morphine, like ethyl alcohol, prevented the onset of pain but failed to exert a modifying influence on the electrocardiographic response to standard exercise.

COMMENT

The findings of this investigation suggest that only 3 of the 14 drugs studied are worthy of continued clinical use as vasodilators in the management of angina pectoris or other states of coronary insufficiency. These drugs are glyceryl trinitrate, papaverine, and pentaerythritol tetranitrate. Glyceryl trinitrate remains the drug of choice for treatment of the acute attack and for prophylaxis just prior to contemplated exertion. For prolonged protection, pentaerythritol tetranitrate (10 to 20 mg., three times a day) appears to excel all other drugs tested. It is superior to papaverine because of the higher proportion of persons influenced favorably, the greater duration of action, and the expense of the latter in effective dosage. When papaverine is prescribed in cases of coronary disease, it would seem that a minimum oral dosage of 3 grains (0.2 gm.) three or four times a day is indicated. Considerably smaller doses, however, are effective parenterally.

The failure of ethyl alcohol to influence the electrocardiographic response to exercise in all patients studied appears to indicate that this agent is without coronary vasodilator action. Alcohol has long been known to be a depressant of the central nervous system capable of decreasing anxiety and promoting a sense of well-being. Since the administration of ethyl alcohol prophylactically may not only create a false sense of physical fitness but remove the protection afforded by angina, the danger of its employment prior to contemplated exertion should be recognized. Consequently, although it effectively relieves anginal pain through a sedative action, the fact that underlying myocardial anoxia remains unaltered by its use must not be overlooked. In these respects, morphine was found to act similarly. Ethyl alcohol appears to have value as a sedative for some patients with coronary disease and seems best taken during the leisurely hours at the end of the day. For the anginal attack, however, it must be considered a poor substitute for glyceryl trinitrate since like morphine it is completely ineffective in preventing or correcting coronary insufficiency.

As Starr [3] has pointed out, generalized vasodilatation cannot be produced without a concomitant increase in blood volume. Since rapid increments of blood volume are not possible, the blood needed to fill vessels the diameter of which has rapidly increased must be withdrawn from other vessels the diameter of which must diminish correspondingly. Vasodilatation in some vascular areas causes reflex vasoconstriction in others so that an exchange of blood flow by a borrowing-lending process would seem operative. With this concept in mind, it is perplexing to find that different authors advocate the same drug for augmenting cerebral, coronary, renal, and peripheral blood flow. The fact that ethyl alcohol causes dilatation of skin vessels and draws blood to the surface of the body would seem to suggest that it may fail to augment coronary blood flow. This was actually substantiated by our studies. Similarly, the recognized value of tolazoline hydrochloride as a dilator of peripheral vessels should raise the question of whether or not this drug might actually decrease coronary blood flow. Several of our observations with this drug have at least pointed in that direction.

Heparin and bishydroxycoumarin have been considered of value by Gilbert and Nalefski [4] in the treatment of coronary disease, because of an alleged action of these drugs in increasing coronary blood flow. Similarly, Graham and associates [5] have reported marked relief of symptoms in patients with angina pectoris who had weekly or biweekly injections of 50 to 100 mg. of heparin. The improvement, however, was attributed by these authors to the profound effect of heparin on lipoprotein metabolism, rather than to a vasodilator or antithrombotic action. Our studies [2b] with these drugs have shown no demonstrable effect on the electrocardiographic response to standard exercise; moreover, we have been unable to confirm the striking subjective improvement in anginal symptoms reported by others from the use of heparin.

SUMMARY AND CONCLUSIONS

Evaluation of vasodilator drugs in the treatment of coronary disease presents a difficult problem because of the lack of objective methods of study, the inapplicability of animal observations to man, the element of subconscious bias, and the inaccuracy of clinical estimates of coronary reserve and myocardial anoxia that are based on the symptom of pain. We attempted to avoid some of these pitfalls by recording and comparing the ability of specific agents to modify the electrocardiographic response to standard exercise (Master two-step test) in carefully selected cases.

The findings seem to justify the following conclusions: 1. Glyceryl trinitrate (nitroglycerin) in therapeutic doses exerts a strikingly favorable effect on the response to exercise as recorded electrocardiographically. 2. Papaverine in the dosage of 1 to 2 grains (0.065 to 0.12 gm.) intravenously or 3 to 8 grains (0.194 to 0.518 gm.) orally is effective in some patients in decreasing or abolishing the abnormal electrocardiographic response to standard exercise. Such benefit was not, however, observed with usual therapeutic doses of the drug. 3. Of all the agents tested, pentaerythritol (Peritrate) tetranitrate

3. Starr, I.: George E. Brown Memorial Lecture: Physiologic Considerations Concerned with the Pathogenesis and Treatment of Obstructive Vascular Disease, Circulation **6**: 643 (Nov.) 1952.

4. Gilbert, N. C., and Nalefski, L. A.: The Effect of Heparin and Dicumarol in Increasing the Coronary Flow Volume, J. Lab. & Clin. Med. **34**: 797 (June) 1949.

5. Graham, D. M., and others: Blood Lipids and Human Atherosclerosis: The Influence of Heparin upon Lipoprotein Metabolism, Circulation **4**: 666 (Nov.) 1951.

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appears to be the most effective drug currently available for prolonged prophylactic therapy in angina pectoris. A 10 to 20 mg. dose affords protection for four to five hours, as judged from the two-step test in the majority of patients studied. 4. Aminophylline, Roniacol (beta-pyridyl-carbinol tartrate), tolazoline (Priscoline) hydrochloride, tetraethylammonium chloride, octyl nitrite, dioxyline (Paveril) phosphate, visammin (khellin), heparin, and bishydroxycoumarin (Dicumarol) appeared unimpressive when evaluated according to this method. 5. Ethyl alcohol, in 1 to 2 ounce amounts, consistently fails to influence the electrocardiographic response to exercise, although it does prevent or reduce the severity of anginal pain. Alcohol should, therefore, be recognized as a rapidly acting sedative and should no longer be regarded as a coronary vasodilator drug. Morphine is identical to alcohol in its effect on pain and in its failure to modify the exercise response. 6. Of the drugs tested, only glyceryl trinitrate, papaverine, and pentaerythritol tetranitrate appear worthy of continued clinical use as vasodilators in the management of angina pectoris.

176 Hart Blvd. (Dr. Russek).

CLINICAL NOTES

CORONARY EMBOLISM

John T. O. Cheng, M.D.
William J. Cahill, M.D.
and
Edmund F. Foley, M.D., Chicago

Coronary embolism is a very rare occurrence. The first case was reported by Virchow [1] in 1856. Saphir,[2] in 1933, collected 16 cases of coronary embolism from the literature and reported 3 that he had observed himself. In 1941, Hamman [3] was able to refer to 40 cases, 30 from the literature and 10 from records of the Johns Hopkins hospital. In 1950, Moragues, Bawell, and Shrader,[4] after reviewing the literature, were able to refer to 45 cases and reported one of their own. Since then, individual cases have been reported by Mussafia,[5] Rodario,[6] Facquet,[7] Ravera,[8] Aguiar,[9] Glushien,[10] and Walker.[11] A total of 53 cases of coronary embolism have been reported in the literature to date. Among them only three were diagnosed during life. We present a case of coronary embolism in which the diagnosis was made before death and confirmed at autopsy.

REPORT OF A CASE

A 21-year-old white woman, gravida 3 and para 2, first entered the hospital on Dec. 12, 1950. She was admitted to the surgical obstetric service complaining of pain in the right flank when the fetus moved and also of easy fatigability. She stated that at 10 years of age she had "St. Vitus' dance" that compelled her to stay in bed for two years because she was unable to walk. After those two years, she resumed her normal activities without any difficulties. In June, 1948, she spontaneously delivered her first child, at which time her physician told her that she had "heart trouble." Her second spontaneous delivery in August, 1949, of a 6 lb. 10 oz. (3,005 gm.) boy was uneventful. After this delivery, the patient's tolerance to exercise gradually decreased. She stated that she became short of breath when climbing stairs, although she complained of no dyspnea at rest, orthopnea, ankle edema, or any other cardiac symptoms. She became pregnant for the third time and was well until two weeks prior to her admission to the hospital, at which time there developed an increase in exertional dyspnea and pain associated with fetal movement. The patient had been receiving no prenatal care, and she was not taking any cardiac medication. The rest of her history was noncontributory except that her mother died of rheumatic heart disease at the age of 31.

Physical examination on admission revealed a young woman who did not appear acutely ill. Her temperature was 98.2 F; pulse 104 per minute; respirations 20 per minute; and blood pressure 105/75 mm. Hg. There was a slight malar flush. Lungs showed moist basilar rales bilaterally. Sinus rhythm was regular. Apical impulse was in the fifth interspace in anterior axillary line. A systolic, blowing murmur and a mid-diastolic rumble with a palpable thrill were found at the apex. A pulmonic systolic murmur with a reduplicated second tone was also heard. P_2 was louder than A_2. The liver was enlarged and extended 3 cm. below the costal margin. Hepatojugular reflex was negative. The uterus contained a 36 to 38-week-old fetus. The legs were not edematous.

A chest film showed the heart to be increased in transverse diameter, with fulness of the pulmonary artery segment. There was also a suggestive double density overlying the heart that was thought to be due to left auricular enlargement. Definite congestive changes in both lungs were noted, although effusion was not present. Urinalysis showed nothing abnormal, and reaction to a Kahn test was negative. An electrocardiogram revealed a right axis deviation and broad, notched, flat-topped P waves in leads 1 and 2. A diagnosis of rheumatic heart disease, mitral stenosis, and regurgitation with decompensation was made.

While in the hospital, the patient was treated with digitalis, ammonium chloride, and meralluride sodium solution (Mercuhydrin). She was kept at absolute bed rest and maintained on a low salt diet. Under this management, compensation was achieved, and, on Jan. 3, 1951, the patient delivered spontaneously. Tubal ligation was performed two days after the delivery. Compensation was good throughout both procedures, and the patient was discharged on Jan. 15, 1951, in good condition.

The patient entered a women's medical ward of the same hospital on Aug. 28, 1952, at 9:25 a. m. as a case of acute cardiac emergency. During the preceding 19 months, the patient had been well, had cared for her family, and had no symptoms of decompensation in spite of the fact that she was taking no cardiac medicaments. She was perfectly well until an hour and a half before admission, when dyspnea, cyanosis,

From the Department of Medicine, Cook County Hospital.

Dr. P. Szantos examined and reported on the pathological material and Dr. M. Franklin assisted in the preparation of this manuscript.

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severe palpitation, and respiratory distress developed. After being seen by a local physician, she was transported to the hospital in a pole ambulance with oxygen.

Physical examination showed an apprehensive, conscious white woman in acute cardiorespiratory distress. She was cyanotic and dsypneic, and her skin was cold and moist. Blood

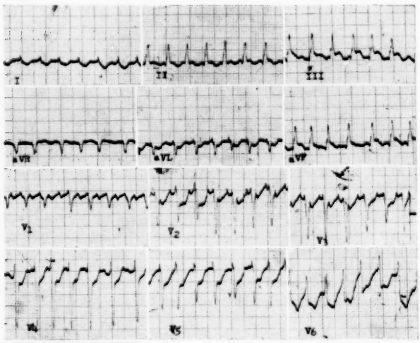

Fig. 1.—Standard limb leads, unipolar limb leads, and unipolar precordial leads showing an acute posterior myocardial infarction.

pressure was 80/10 mm. Hg; temperature 98.5 F; respirations 40 per minute; and pulse thready and irregular, about 48 to 50 per minute. Neck veins were engorged. Lungs were full of inspiratory and expiratory rales and coarse rhonchi. The patient was frothing at the mouth. Apical heart rate was 180 per minute and irregular; it was too rapid to detect any adventitious sounds. A marked precordial heave was noted. The abdomen was soft and not distended. The liver was felt 3 cm. below the costal margin; its edge was round; and the organ was only slightly tender. The extremities were cold and clammy but not edematous. Urinalysis revealed a specific gravity of 1.014, no albumin, no sugar, and 1 to 2 white blood cells per high power field.

The patient was given 95% alcohol aerosol in oxygen immediately on admission and, also, aminophylline, 7 grains (0.45 gm.), and digitalis (Digalen), 3 grains (0.19 gm.), intravenously.

Fig. 2.—Thickened and deformed cusps of the mitral valve and the mural thrombus in the left auricle.

An electrocardiogram was made. It showed auricular fibrillation, with a rapid ventricular response, prominent Q waves in leads 2, 3, and aVF, elevated S-T segments in leads 2, 3, and aVF, and reciprocal depression of S-T segments in leads 1, aVL, V_2, V_3, V_4, V_5, and V_6 indicative of an acute posterior myocardial infarction (fig. 1). A diagnosis was made of recent myocardial infarction of the posterior wall caused by coronary embolism resulting from a thrombus formed in a fibrillating left auricle in a rheumatic heart with mitral stenosis.

The patient's course was rapidly downhill. She was given another 3 grains of digitalis intravenously. Heart tones became imperceptible. An intracardiac injection of 2 cc. of 1:1,000 solution of epinephrine reinstituted an irregular cardiac beat that persisted for two to three minutes until apnea began and then faded. Despite all therapy, the patient died at 10:30 a. m. on Aug. 28, 1952.

At autopsy, the body was cyanotic, and the chest was somewhat emphysematous. Each pleural cavity contained approximately 2,000 cc. of serous fluid, and the pericardial cavity contained 80 cc. of serous fluid.

The heart weighed 350 gm. and was markedly enlarged. Apex and base diameters were 12 cm., and transverse diameter at the base was 10 cm. The epicardium was smooth and glistening, and there was a normal amount of subepicardial fat tissue. The myocardium of the right ventricle was 3 mm. thick and that of left ventricle 14 mm. The tricuspid valve measured 13 cm. in circumference and was not abnormal. Pulmonic valves measured 8 cm. in circumference and were normal. Mitral valve measured 5 cm. in circumference. The valve leaflets were markedly thickened, with shortening and thickening of papillary muscles and chordae tendinae (fig. 2). The aortic valve measured 8 cm.; the cusps were smooth and normal. There was little atherosclerosis of the aorta. The left auricle was greatly dilated and hypertrophied. In the left auricular appendage, a thrombus was noted (fig. 2). The coronary vessels

Fig. 3.—Embolus in the circumflex branch of the left coronary artery.

showed no arteriosclerotic changes. In the left circumflex branch of the left coronary artery, an embolus measuring 4 by 2 by 2 mm. was seen that completely occluded the lumen of this vessel (fig. 3). The embolus was laminated and was not adherent to the vessel wall. The myocardium of the posterior wall of the heart showed a large area of tissue that appeared paler and drier than normal.

Both lungs weighed 1,300 gm. and were heavier than normal. Cut section revealed a large amount of frothy, sanguinous fluid. The pulmonary arteries were clear of thrombi. The liver weighed 1,600 cm., showed considerable blunting of the inferior margin, and was severely congested. The spleen weighed 300 gm. and was markedly congested. The only finding in the esophagus, stomach, and small and large bowels was congested mucosal surfaces. The adrenal glands were normal. Kidneys weighed 300 gm. The capsule stripped with ease and revealed a smooth cortical surface; a cut section showed evidence of congestion. The uterus was that of a multipara and was normal. Both tubes were cut and ligated in the isthmic portion. The left ovary contained a corpus luteum. The brain weighed 1,000 gm. and was normal.

Microscopic section from the area of infarction in the left ventricle revealed fragmentation of the myocardium. The nuclei of some of the fibers were pyknotic; the cross striations, however, were still preserved. The interstitial connective tissue was edematous. The capillaries and thin-walled veins were markedly engorged.

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COMMENT

Embolic occlusion of the coronary arteries is a rare occurrence. The infrequency of this condition has been attributed to (*a*) the difference between the caliber of the aorta and that of the coronary, (*b*) the situation of the coronary vessels at the root of the aorta, (*c*) the right angle emergence of the coronary arteries, (*d*) the bulk and swiftness of the blood current in this portion of the aorta, and (*e*) the fact that the major part of coronary filling occurs during diastole. In reviewing the literature, there appear to be eight possible sources of emboli that may occlude the coronary arteries. The sources of emboli in the cases reviewed in the literature are listed in the table. In our patient, the embolus originated in the left auricular appendage and was sterile.

According to Garvin,[12] in 25% of all cases of heart disease, there are mural thrombi in the left side of the heart, which predispose it to the occurrence of embolism. Small emboli cause no conspicuous symptoms. Only

Causes of Emboli in Cases of Coronary Embolism Reported in the Literature

Author	Bacterial Vegetations on Mitral or Aortic Valve Due to Bacterial Endocarditis	Mural Thrombi on Arteriosclerotic or Syphilitic Lesion in Aorta	Intraventricular Mural Thrombi	Paradoxical Embolism (Thrombi in Peripheral Veins)	Thrombus or Atheromatous Material in Coronary Artery	Thrombi in Pulmonary Veins	Air Embolism	Calcified Aortic Valve
Mussafia [5]	1	..	..	..	..	..	..	..
Moragues et al.[4]	20	10	5	4	2	2	..	1
Facquet et al.[7]	..	..	..	..	..	..	1	..
Ravera [8]	1	..	..	..	..	..	..	..
Aguilar et al.[9]	..	..	..	..	1	..	..	..
Ghishlen et al.[10]	1	..	..	..	..	..	..	..
Walker [11]	1	..	..	..	..	..	..	..
Totals	24	10	5	4	3	2	1	1

when they are large enough to occlude big arteries or lodge in organs in which even relatively small infarcts produce grave symptoms does their presence become noticeable. The two organs most sensitive to infarction are the brain and the heart. In cardiac patients, evidence of cerebral embolism occurs commonly; coronary embolism occurs infrequently. When the peculiarities of the coronary circulation as previously enumerated are considered, infrequent occurrence of the latter is not remarkable.

Coronary occlusion due to embolism not only is a very rare occurrence but is rarely diagnosed during life. In only three [13] cases was the correct diagnosis made during life; all three were cases of bacterial endocarditis; in all three, the emboli were not sterile; and all three patients lived long enough to reveal distinctive clinical and electrocardiographic features.

Our case was unique in several respects. The embolus originated in a thrombus in left auricle, and bacterial endocarditis was not present. Both the thrombus and the embolus found in the coronary artery were sterile. The coronary embolism involved the circumflex branch of the left coronary, a site of occlusion by embolus hitherto not reported in the literature. Our patient lived only a little more than one hour, the shortest survival time among cases of coronary embolism that were correctly diagnosed before death.

We reconstructed the sequence of events in our case as follows. The enlargement of the left auricle followed mitral stenosis, and, as frequently happens, a thrombus formed in the cavity of left auricle, particularly in the appendage. Also, auricular fibrillation might have contributed to the formation of the auricular thrombus. From the thrombus in the left auricle came the embolus that lodged in the circumflex branch of the left coronary artery and that caused posterior myocardial infarction, acute left ventricular failure, and death. Although the myocardial infarction was evident grossly, we failed to demonstrate microscopically any definite necrosis of the myocardium in the infarcted area. Apparently the patient succumbed to the shock of acute occlusion of the coronary artery so soon that there was no time for tissue necrosis to become evident. In patients who die within 12 hours after a coronary occlusion, histological evidence of infarct in the heart may not be found.[14]

Although embolic occlusion of the coronary arteries is very uncommon, it is probably commoner than the few reports in the literature would indicate.[3] Hamman [3] conservatively estimated that from 1 to 2% of the cases of coronary occlusion are due to embolism; therefore, it is reasonable to assume that only a small number of the cases encountered at autopsy have been reported. When symptoms are minimal or atypical, coronary embolism is seldom suspected, and, if the patient survives, especially a person in the older age group, the diagnosis of occlusion caused by embolism cannot be proved. How often recovery occurs after coronary embolism is not known; however, probable instances have been reported.[15] It is predicted that the diagnosis of coronary embolism will be made more frequently as interest and familiarity incite more thorough investigation into each case of coronary occlusion, especially in young persons.

SUMMARY

A case of coronary embolism is reported, and a brief review of the literature is presented. This is the fourth case of coronary embolism that was diagnosed while the patient was alive. This patient had rheumatic heart disease, with mitral stenosis. Bacterial endocarditis was not present, and the source of the embolus was a left auricular thrombus. The occluded coronary artery was the left circumflex branch.

1835 W. Harrison St. (12) (Dr. Cheng).

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Fibrosis of the Sphincter of Oddi.—Obstruction to the free flow of bile through the lower end of the common duct due to lesions of the ampulla, the papilla of Vater, and the sphincter of Oddi occurs in a sufficient number of patients to make this a problem of real clinical importance. This obstruction can occur with or without pathologic conditions elsewhere in the extrahepatic biliary tract but is usually encountered in patients with gallstones. This intermittent or persistent obstruction is responsible for an appreciable number of the unsatisfactory results following cholecystectomy for cholelithiasis.—Richard B. Cattell, M.D., and Bentley P. Colcock, M.D., *Annals of Surgery*, June, 1953.

RUPTURE OF THE AORTIC VALVE

DISAPPEARING DIASTOLIC PRESSURE AS A DIAGNOSTIC SIGN

William B. Bean, M.D.
and
Martha C. Schmidt, M.D., Iowa City

Rupture of the aortic valve falls into two distinct clinical patterns; the first is rupture of the normal valve after some unusual stress or strain, particularly that of heavy lifting [1]; the other occurs when the valve is disorganized by bacterial endocarditis and erosion leads to rupture.[2] We wish to present an example of the second kind and emphasize a physical sign that may be helpful in detecting the aortic rupture clinically; namely, a sudden fall or complete disappearance of the diastolic blood pressure. Clinical signs of established aortic insufficiency are well known, but the observation of a normal diastolic pressure that declines significantly or disappears entirely has not been emphasized.

REPORT OF A CASE

L. C., a 44-year-old man, was admitted to the medical service of the University Hospitals because of chest pain and fever. Four weeks before admission he had a toothache and a swollen upper lip. Before he was able to consult a dentist, he was seized with a shaking chill, and his temperature rose to 104 F. His physician gave him 400,000 units of penicillin daily for a 10 day period. During this time the patient felt very much better, but, in spite of treatment, he had another chill and a fever of 105 F a week after the first chill. During the three week period after the first chill, he had four more similar episodes. He was then admitted to the local hospital. Laboratory studies revealed a hemoglobin level of 67%, red blood count of 4 million and sedimentation rate of 44 mm. per hour. Agglutination tests for typhoid and brucellosis were negative. Three days before he was transferred to the University Hospitals, the patient became orthopneic and had precordial pain that radiated to the back and increased on inspiration. The day before admission he noted a tender, red, swollen area approximately 3 cm. in diameter on the dorsum of the right foot. Sulfadiazine had been given along with penicillin.

On the patient's admission to the University Hospitals, physical examination revealed a seriously ill, orthopneic, dyspneic man who was perspiring freely. The neck veins were slightly engorged. There was decrease in resonance and tactile fremitus over the axilla on the left. Breath sounds were distant, with wheezes throughout both lungs and numerous inspiratory rales at both bases. The heart was enlarged, the left border extending to the anterior axillary line. A soft systolic and a grade 2 diastolic murmur were heard at the apex. There was an occasional extrasystole, but otherwise the heart rhythm was regular. The liver, slightly tender, was palpable two inches below the costal margin on the right. The erythematous area on the dorsum of the right foot was noted. Temperature was 104 F, pulse 104, respirations 20, and blood pressure was 120/58 mm. Hg. The history revealed that the patient had had migratory arthritis when he was 27 years old, and, 10 years prior to admission, he had been rejected for military duty because of "two leaky heart valves."

Laboratory studies revealed a hemoglobin of 8.8 gm. per 100 cc., red blood cell count of 3.2 million per cubic millimeter, and white blood cell count of 13,200 per cubic millimeter, with 74% granulocytes. Sedimentation rate was 64 mm. per hour. The urine had a specific gravity of 1.013 and 1+ albumin. The sediment contained numerous sulfadiazine crystals. A roentgenogram of the chest showed the heart to be enlarged both to the right and the left, with pulmonary vascular congestion. A small pleural effusion was seen in the left costophrenic angle. The electrocardiogram showed auricular fibrillation. Daily blood cultures were obtained, and all were reported subsequently as negative for micro-organisms. The patient was digitalized and put on a regimen of penicillin and oxytetracycline (Terramycin). On the fifth hospital day, petechiae appeared on the soft palate, and a grade 3 precordial friction rub appeared, obscuring the murmurs. The heart rate slowed, and the patient appeared better, but he continued to have a fever of 102 F daily. Oxytetracycline therapy was discontinued, and streptomycin therapy was begun. We suspected that active rheumatic heart disease might be present in addition to subacute bacterial endocarditis. On the 11th hospital day, the patient complained suddenly of pain in the chest. Examination revealed the pulse to be 92 and the blood pressure 170/10 mm. Hg. Broadbent's sign was present. By the next day the heart had enlarged greatly, and dyspnea was an increasingly troublesome symptom. The pericardial friction rub was louder and obscured the underlying murmurs over the entire precordium. Pulsations of the carotid and subclavian arteries became prominent. Corrigan's pulse, capillary pulsation, and pistol-shot sounds appeared. A roentgenogram of the chest revealed great increase in the enlargement of the cardiac silhouette, in addition to increasing pulmonary congestion (fig. 1). Veins in the neck were distended, and the roentgenologic studies were consistent with pericardial effusion. A pericardial tap yielded 180 cc. of bloody fluid that had a specific gravity of 1.015 and contained 3,300 leukocytes. Corticotropin, 150 mg. daily, was added to the treatment program. The next day the pericardial friction rub, though still loud, was confined to a 3 cm. area at the level of the fifth intercostal space to the left of the sternum. The fever continued. On the 14th hospital day, the blood pressure was 140/0 mm. Hg, and there was no clinical change. On the next day, four days after the sudden drop in the diastolic pressure, the patient suddenly went into peripheral vascular collapse and died. A clinical diagnosis of rupture of the aortic valve was made, and it was suspected that, in addition to bacterial endocarditis, there might have been active rheumatic fever.

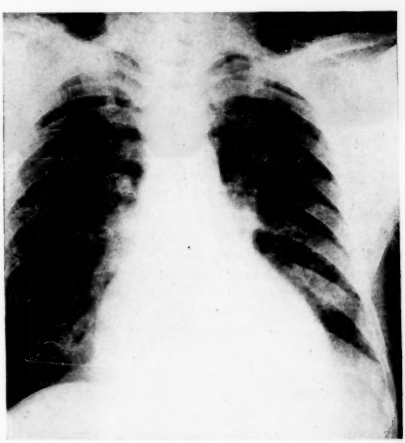

Fig. 1.—Roentgenogram of the chest on the patient's admission to University Hospitals.

Autopsy revealed the pericardial sac to be distended with approximately 2,000 cc. of grossly bloody fluid. The heart weighed 900 gm. and was covered with dense, recent, fibrinous

From the Department of Internal Medicine of the College of Medicine, State University of Iowa, and the University Hospitals.

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adhesions. The mitral valve leaflets were thickened and adherent along the margin, the valve orifice admitting only the tip of the index finger. The aortic valve cusps were irregularly thickened and partly calcified. The anterior cusp was the most seriously diseased. The ventricular surfaces of the anterior cusp and the left posterior cusp were covered with friable blood clot and debris. The body of the anterior cusp was torn with a ragged defect approximately 7 mm. in diameter. There was another perforation through the anterior and the adherent

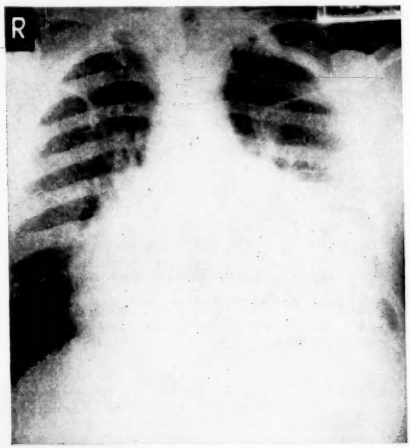

Fig. 2.—Roentgenogram of the chest one day after rupture of the aortic valve, before removal of fluid from the pericardium.

left posterior cusp that entered an opening between the sinuses of these cusps. There was a defect in the intima of the sinus of Valsalva of the anterior cusp that did not extend through the aortic wall. No gross communication could be found between the lumen of the aorta and the pericardial cavity. The left lower lobe of the lung was atelectatic. The liver and spleen were congested. The left kidney and the spleen contained several infarcts. Microscopic sections of the heart showed evidence of old rheumatic pancarditis. The vegetations of the aortic valve contained colonies of bacteria. Postmortem cultures from the heart valve yielded Pseudomonas aeruginosa and nonhemolytic streptococci.

COMMENT

The diagnosis of rupture of the aortic valve in the course of subacute bacterial endocarditis has been a difficult clinical problem. The complexity is increased by the notorious variability of murmurs, which change with distortion or dilatation of the valve and the vegetation. Curious murmurs may come and go. The change of a murmur or the occurrence of a new murmur, therefore, is not a sure sign of aortic valve rupture. More emphasis has been given to the abrupt onset of pain in the chest. This was a prominent symptom in our patient, but its possible connection with pericarditis was hard to evaluate. Likewise, the appearance or increase of congestive failure is a helpful sign, and this was dramatic in our patient. But the most significant clinical change was the fading away of diastolic arterial pressure and the appearance of all the peripheral signs of aortic valve leak—Corrigan's pulse, capillary pulsation, and pistol-shot sounds. The sudden appearance of these signs with the advent of aortic regurgitation casts some doubt on Lewis' interpretation of such signs as a reflection of peripheral vasodilatation rather than central reflux.[3] At least the signs first appeared when the valve ruptured and the diastolic pressure vanished.

The great outpouring of bloody pericardial fluid could not be shown to have followed a dissection or rupture through the coats of the aorta or through the heart muscle. This possibility exists. Undoubtedly, congestive failure contributed some transudate. Anderson, Christenson, and Edwards have demonstrated hemopericardium of the myocardial infarction without rupture when "bleeding arose from pericarditis in areas removed from the infarcted myocardium."[4] Such a process may have been responsible in this case.

Is there any use in being able to discover rupture of the aortic valve during life, other than for the clinician's pride? That the diagnosis is truly difficult is indicated by the fact that only one of Carroll's 15 cases was recognized before autopsy.[2] The time may come when surgeons will be able to repair such terrible damage and save the patient.

SUMMARY

A case of rupture of the aortic valve, diagnosed before death, is reported. The abrupt decline and disappearance of the diastolic arterial blood pressure should be emphasized along with sudden pain, the advent or increase in congestive failure, and the appearance or change of an aortic diastolic murmur, as an important diagnostic sign of rupture of a diseased aortic valve in bacterial endocarditis.

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DISSEMINATED LUPUS ERYTHEMATOSUS WITH INVOLVEMENT OF THE SPINAL CORD

Philip G. Piper, M.D., Madison, Wis.

A review of the literature on involvement of the central nervous system in disseminated lupus erythematosus revealed no case of focal involvement of the spinal cord in addition to the other protean manifestations. Since Hebra and Kaposi [1] and Bowen [2] described mental changes associated with this disease, Daly [3] and Sedgwick and Von Hagen [4] have more completely summarized the clinical and pathological findings. They reported toxic delirium and coma in association with the anatomic alterations of diffuse nonspecific encephalitis, as well as

From the Department of Pathology, University of Wisconsin Medical School.

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vascular changes and thrombosis producing myelomalacia of the nervous parenchyma secondary to disturbances in the vascular supply. The following case is reported because of the unusual finding of vascular changes with thrombosis of the meningeal vessels resulting in ischemic necrosis of the lumbar cord.

REPORT OF A CASE

A 19-year-old white girl who had disseminated lupus erythematosus was admitted to the University Hospitals for the sixth time on May 3, 1952. She had responded fairly well to cortisone therapy until an exposure to the sun in April caused an exacerbation of the "butterfly" rash, joint pains, and fever. Immediately prior to hospitalization she experienced numbness and tingling of both lower extremities, which gradually extended up to the trunk with progressive weakness, marked diminution in motility, and difficulty with urination.

Physical Findings.—Physical examination revealed an acutely ill patient with a maculopapular erythematous rash of "butterfly" pattern over the face. Transient rhonchi were noted occasionally on the right side. The liver was palpated 2 cm. below the right costal margin, and the spleen 1 cm. below the left costal margin. The reflexes were generally hypoactive. There was considerable loss of sensitivity to pin prick in the perianal area, and a Babinski reaction was elicited on the left.

Treatment had consisted of 50 mg. of cortisone daily, which was later increased to 75 mg. to control intermittent fever. On admission the patient was given 300,000 units of crystalline procaine penicillin (Duracillin) twice daily to prevent possible infection and was given corticotropin by continuous intravenous drip. Several days later crystalline procaine penicillin and *p*-aminobenzoic acid, 3 gm. each, three times daily, were given. There was no response to therapy. The fever continued, and the patient became progressively worse. She became affected with generalized twitching and died on June 17, 1952.

Fig. 1.—Focal swelling with infarction in the lumbar spinal cord evident on reflection of the dura.

Laboratory Data.—A positive lupus erythematosus phenomenon, albuminuria, persistent leukopenia, and normal prothrombin level were found. Serum proteins were 7.1 gm. per 100 cc., with 3.6 gm. of albumin per 100 cc. and 3.5 gm. of globulin per 100 cc. The cerebrospinal fluid protein was 175 mg. per 100 cc., blood sugar 39 mg. per 100 cc., and serum chloride 106 mEq. per liter. There were 16 white cells, most of which were lymphocytes. Result of the colloidal gold test was 5555432100, and the Wasserman reaction was negative. The Queckenstedt reaction was normal. The L. E. cells were identified by the Haserick [5] phenomenon. The mature neutrophilic leukocytes contained within the cell membrane one or more masses of nuclear material, which stained a bluish-purple color. These masses are theoretically composed of a depolymerized desoxyribose nucleic acid. The formation of the L. E. cell occurs when the blood is exposed to air outside of the body, in conjunction with the gamma globulin of the plasma of the patient. Since approximately 96% specificity can be correlated with the finding of the L. E. cell, the phenomenon in this case supported the diagnosis.

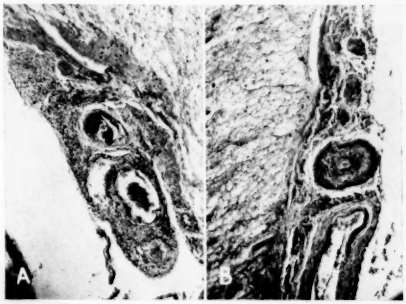

Fig. 2.—*A*, vasculitis of the meningeal vessels in the area of spinal cord swelling, with fibrinoid deposition in the vessel wall and degenerative change of the adjacent cord (× 100). *B*, a recanalized thrombus within a meningeal vessel adjacent to the area of spinal cord myelomalacia (× 100).

Autopsy Findings.—At necropsy the patient appeared older than her stated age and had a 3 + pitting edema of the lower extremities. The abdomen was distended and tympanitic. No rash was evident on the face.

When the thorax was opened, multiple fibrous adhesions were found between the visceral and parietal pleura of both chest cavities. The heart weighed 250 gm. and presented at the point of attachment of one chorda tendinea of the mitral valve a white area of discoloration, which on microscopic examination revealed a focal myocarditis filled with many inflammatory cells and scattered clusters of cocci. On culture these were identified as Staphylococcus aureus. The remainder of the heart was essentially normal. Both the right and left lungs were approximately twice normal weight. The lungs revealed areas of atelectasis, and on section a considerable amount of fluid was expressed. The cut surface revealed a confluent bronchopneumonic process involving the middle and lower right lobes and the lower left lobe.

The spleen was enlarged and weighed 500 gm. The surface presented multiple infarcts, the largest measuring approximately 3 cm. across. The pulp was firm and nonfriable, and the Malpighian corpuscles were readily identified. On microscopic examination the areas of infarction contained clusters of cocci. The liver weighed 1,750 gm. and was grossly normal as were the gallbladder, pancreas, alimentary tract, and adrenals. The kidneys each weighed 250 gm. Capsules stripped with difficulty, and the cut surfaces presented a grayish-white, mottled appearance. Small discrete foci of necrosis were identified over the surface and deep within the kidney parenchyma. Microscopic examination of these areas revealed cocci. A minimal thickening of the basement membrane of the capillaries was occasionally observed. Foci of hemorrhage were also seen. An occasional crescent was found over the glomerules in the Bowman's capsule. No "wire loop" configurations were identified within

5. Haserick, J. R.: Plasma L. E. Test in Systemic Lupus Erythematosus, J. A. M. A. **146**: 16-20 (May 5) 1951.

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the glomerular capillaries. The ureters and bladder were normal. The pelvic organs were those of a normal woman.

Examination of the brain revealed no signs of increased intracranial pressure. The pons, medulla, and cerebellum showed no gross lesions. On microscopic examination, however, the meninges were slightly thickened. The vessels were hyperemic, and certain vessels presented an acute vasculitis with perivascular demyelinization. Some of the vessels showed fibrinoid change within the wall. There were scattered petechial hemorrhages within the right basal ganglion. Examination of the spinal cord revealed a fusiform swelling in the lumbar area that measured 3 cm. in length and 1.5 cm. in diameter through the thickest portion (fig. 1). On microscopic examination through this area, the meningeal vessels presented thickening of walls, perivascular infiltration, and fibrinoid change within the walls (fig. 2*A*). All the vessels surrounding the area of enlarged cord contained thrombi, and one appeared to be recanalized (fig. 2*B*). The posterior funiculi showed myelomalacia. The anterior horns appeared intact, but there was a loss of ganglion cells in the posterior horns.

COMMENT

Since the cultures taken from the septic splenic infarcts, the kidneys, and from the area of discoloration of the chorda tendineae of the mitral valve all revealed Staphylococcus aureus, it was felt that the patient died from a fulminating staphylococcic septicemia. The lack of the characteristic findings of Libman-Sacks verrucae, "wire loop" glomerular capillaries, alteration of connective tissue of the cutis, and the fibrinoid degeneration of dispersed connective tissue of the body may well be attributed to the prolonged cortisone therapy.

Previous cases have been reported in which the essential lesion was vasculitis with fibrinoid change of the involved vessels and an associated myelomalacia and which occasionally would give rise to adhesive meningitis or chronic meningoencephalitis. It was considered that the brain lesions associated with vasculitis were incidental rather than the primary cause of death, except in those instances when gross infarction or hemorrhage occurred. Although the vessels within the brain proper and the meninges were involved, the most significant alterations in this case were in the blood supply to the spinal cord. Because of the multiple splenic infarcts, the possibility of infarction secondary to embolization of the vessels to the cord was considered; however, in this case there was no source for such an embolus. Ross and Wells [6] have reported, in a survey of the literature, that in approximately 28% of patients who died with disseminated lupus erythematosus, multiple splenic infarcts were found with no demonstrable source of emboli. The complete diagnosis was systemic staphylococcic septicemia with multiple focal septic abscesses; spinal cord swelling and degeneration due to vasculitis with thrombosis of the vessels supplying the meningeal vessels to the lumbar cord; cerebral hyperemia and minimal vasculitis; bilateral bronchopneumonia; pulmonary edema; arteriolonephrosclerosis; splenomegaly; and multiple septic splenic infarcts.

SUMMARY

A case is reported as an illustration of a rare complication of disseminated lupus erythematous, namely, vascular involvement of the spinal cord producing an infarct with resulting paralysis of the lower extremities.

426 Charter St.

6. Ross, W. S., and Wells, B. B.: Systemic Lupus Erythematosus: A Review of the Literature, Am. J. Clin. Path. **23**: 139-160, 1953.

COUNCIL ON FOODS AND NUTRITION

ACCEPTED FOODS

The following products have been accepted as conforming to the rules of the Council.

JAMES R. WILSON, M.D., *Secretary.*

Webster-Thomas Company, Boston.

Matchless Brand Dietetic Pack Apple Sauce.

Ingredients: Apples processed and packed in water without any added sugar or salt.

Analysis (submitted by distributor).—Total solids 11.3%, moisture 88.7%, fat (ether extract) 0.13%, protein (N × 6.25) 0.15%, crude fiber 0.57%, available carbohydrate 9.71%, sodium 1.6 mg./100 gm.

Calories.—0.41 per gram; 11.7 per ounce.

Use.—For use in low sodium and other therapeutic diets.

Matchless Brand Dietetic Pack Cut Green Beans.

Ingredients: Tendergreen variety beans packed in water without any added salt.

Analysis (submitted by distributor).—Total solids 5.2%, moisture 94.8%, fat (ether extract) 0.11%, protein (N × 6.25) 0.88%, crude fiber 0.53%, available carbohydrate 2.92%, sodium 1.02 mg./100 gm.

Calories.—0.17 per gram; 4.8 per ounce.

Use.—For use in low sodium and other therapeutic diets.

Matchless Brand Dietetic Pack Green Lima Beans.

Ingredients: Thorogreen variety baby lima beans packed in water without any added sugar or salt.

Analysis (submitted by distributor).—Total solids 19.5%, moisture 80.5%, fat (ether extract) 0.28%, protein (N × 6.25) 5.80%, crude fiber 1.58%, available carbohydrate 9.51%, sodium 10.32 mg./100 gm.

Calories.—0.64 per gram; 18 per ounce.

Use.—For use in low sodium and other therapeutic diets.

Matchless Brand Dietetic Pack Cut Wax Beans.

Ingredients: Kinghorn variety wax beans packed in water without any added salt.

Analysis (submitted by distributor).—Total solids 4.6%, moisture 95.4%, fat (ether extract) 0.09%, protein (N × 6.25) 0.82%, crude fiber 0.54%, available carbohydrate 2.37%, sodium 0.80 mg./100 gm.

Calories.—0.13 per gram; 3.7 per ounce.

Use.—For use in low sodium and other therapeutic diets.

Matchless Brand Dietetic Pack Sliced Beets.

Ingredients: Detroit dark red beets packed in water without any added salt.

Analysis (submitted by distributor).—Total solids 8.0%, moisture 92.0%, fat (ether extract) 0.05%, protein (N × 6.25) 0.90%, crude fiber 0.53%, available carbohydrate 5.53%, sodium 46.50 mg./100 gm.

Calories.—0.26 per gram; 7.3 per ounce.

Use.—For use in low sodium and other therapeutic diets.

Matchless Brand Dietetic Pack Diced Carrots.

Ingredients: Red cored chantenay carrots packed in water without any added salt.

Analysis (submitted by distributor).—Total solids 6.56%, moisture 93.44%, fat (ether extract) 0.06%, protein (N × 6.25) 0.78%, crude fiber 0.60%, available carbohydrate 4.04%, sodium 50.1 mg./100 gm.

Calories.—0.20 per gram; 5.7 per ounce.

Use.—For use in low sodium and other therapeutic diets.

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X-RAYS DURING PREGNANCY

Contemporary radiobiological research has stimulated interest in and controversy over the magnitude of the radiation hazard associated with roentgen pelvimetry and other diagnostic procedures that expose the fetus to x-rays. Many aspects of the problem were discussed at a recent conference on the effects of radiation and other deleterious agents on embryonic development, sponsored by the biology division of the Oak Ridge National Laboratory, the proceedings of which will be published soon by Oak Ridge. Very little is known about the nature of whole-body radiation injury in the case of the fetus. It is probable that fetal blood-forming organs and other tissues are just as radiosensitive as adult structures and that comparable physiological disturbances occur. The present discussion is concerned only with genetic effects, particularly as they are manifested in the later development of the individual. Exposure of the fetus to sublethal doses of ionizing radiation may have two important genetic sequelae. These are gene mutations and chromosome aberrations that lead to somatic mutations and result in abnormal development of one or more of the organ systems.

The problem of the production of gene mutations is extremely complex, since they are changes in the heredity of the organism that become apparent only in the next or subsequent generations. Competent authorities differ widely in their estimates of the dose of radiation required to double the natural mutation rate per generation in man. Most of the estimates are based on studies of the fruit fly, Drosophila, and of mice extrapolated in terms of what is known about human genetics. Robley Evans' calculations suggest that doses of the order of 250 to 300 r are necessary to produce this effect. Other workers, such as Sewall Wright, H. J. Muller, D. R. Charles, and L. B. and W. L. Russell, using somewhat different assumptions, have presented estimates that are lower than Evans' by as much as a factor of 100. Nearly all the students of the subject share the belief that there is no threshold dose for gene mutation that may follow any radiation exposure however small. It should be emphasized, however, that the generally accepted level of significance for this genetic effect is a doubling of the normal or spontaneous rate of gene mutation.

Of greater practical importance is the occurrence of somatic mutations that lead to developmental defects. Since these changes are the results of chromosome injury, and since it is known that healing, or restitution, of chromosomes occurs, there should be a threshold dose for this phenomenon. This dose is not known with certainty for any species, but, in the case of one mammal, the mouse, it may be as low as 25 r. This problem has two aspects that are of clinical interest: first, the dose of radiation required and, second, the time during gestation at which it is delivered. The clinical literature contains many reports of the occurrence, and absence, of congenital malformations in children who were irradiated in utero. These reports have seemed to be contradictory and difficult of interpretation until the recent extensive animal experiments of J. G. Wilson, S. P. Hicks, and the Russells were reported. All the contemporary animal studies are consistent, and the results may be summarized conveniently by quoting from a paper of the Russells: "The developing embryo of a great variety of animal forms, including several mammals, is highly susceptible to the induction of malformations by radiation. There is no reason to doubt that this also applies to the human embryo . . . doses high enough to produce developmental abnormalities do not necessarily cause abortion or prenatal death. . . . Animal experiments have clearly demonstrated that there are well defined critical periods in the development of most characters. That is, a particular abnormality may be produced with a high incidence by irradiation at a particular stage or stages, but not at all from irradiation at other stages." [1]

The most important human material that can be compared with animal studies are less than 100 Japanese children who were exposed in utero to the detonation of the atomic bomb. Examination of these children has not revealed an abnormal incidence of skeletal abnormalities; however, the Atomic Bomb Casualty Commission has reported that, of 11 Hiroshima children who were exposed in utero to amounts of gamma radiation that approximated the median lethal dose, 7 were found to have microcephaly with mental retardation, 2 were mongolian idiots, and 2 were apparently normal. The total number of stillbirths and the number of abortions that occurred is not known with certainty, although it is reported that most pregnant women miscarried soon after the incident.

The problem of the critical time is of great interest clinically, since, to quote the Russells again: "Critical periods for the majority of gross abnormalities in mice (and also in rats) occur at a time which, in man, corresponds to the second to the sixth week of gestation. . . . Irradiation at more advanced stages in the mouse (corresponding to periods following the sixth week in man) produces less obvious and possibly more delayed effects which from the human point of view may be as harmful as the gross monstrosity." From their own studies the Russells concluded: "Whenever possible irradiation involving the uterus of women of child-bearing age should be restricted to the two weeks following the last menstrual period to preclude the possibility of fertilization having taken place. This applies particularly to diagnostic irradiation even if the doses involved are less than 25 r."

1. Russell, L. B., and Russell, W. L.: Radiation Hazards to Embryo and Fetus, Radiology 58: 369 (March) 1952.

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There does not appear to be any rational basis for disputing the conclusion that radiation is harmful to the fetus. The difficult problem is the statement of the amount of radiation meant when speaking of harm. From the standpoint of the geneticist who is concerned with gene mutations, any amount in excess of the natural background radiation is capable of inflicting genetic injury. The geneticist, therefore, is entirely consistent when he recommends that the fetus be protected from all radiation. Such a complete interdiction is not practical clinically, and occasions arise when it is necessary to perform roentgenologic examinations of the pelvic region because of injury, suspected dystocia, and the like. Under such circumstances an acknowledged risk is, and should be, accepted. The hazard can be minimized by the use of efficient radiographic equipment and by restricting the examination to the greatest extent compatible with obtaining the desired information. The animal experimentation provides a scientific basis for recommending that those examinations that can be deferred should be performed as late as possible in the course of pregnancy. Since the radiation exposure during fluoroscopy is usually many times greater than during radiography, fluoroscopic examinations of the pelvic region (such as of the colon or small intestine) should not be done at all during pregnancy. Since many office fluorosopes are poorly protected and seldom calibrated and are operated frequently at very high outputs it would be wise for physicians who are not qualified radiologists to defer all fluoroscopic examinations until after delivery. When the characteristics of the machine are well known and when the operator is certain that there are no x-ray leaks, cautious and brief studies of the chest and upper gastrointestinal tract should not be dangerous. The fact remains, however, that it is not possible to specify a dose of x-rays that is "safe" in the sense that it is known to be incapable of causing fetal injury.

The Russells have made an interesting suggestion to reduce the possibility of accidental radiation injury to the very young fetus. The most susceptible period in human embryogenesis appears to be between the second and sixth week of gestation, when few women are aware of the existence of pregnancy. Extensive fluoroscopic or radiographic examinations at this critical time could certainly inflict serious, unintentional injury. It is in x-ray pelvimetry and fetometry that the question of possible radiation damage has been most stressed, but, actually, from the standpoint of the number of patients involved and the doses delivered, examinations such as pyelograms, gastrointestinal examinations, and particularly the examination of the lumbosacral spine are the ones that are more apt to be dangerous. There is a sound experimental basis for the recommendation that all x-ray examinations or treatments involving the uterus should be restricted to the two weeks immediately following the end of a menstrual period. There is a good possibility that conscientious radiologists will adopt this procedure, since it involves no more than a scheduling of certain examinations. Some physicians may object to it as being excessively cautious and may even feel that their patients will become unnecessarily apprehensive about all x-ray exposures. The scientific evidence, however, demonstrates clearly that there is a basis for apprehension and that caution is justified.

MUSIC AS A THERAPEUTIC MODALITY

The use of music as a therapeutic modality is the subject of a recent communication by Arrington,[1] who declares that in acutely ill patients music often produces effects of sedation and reassurance, while in chronically ill patients music is able to divert the mind during long periods of convalescence and enforced rest. Patients with tuberculosis, rheumatic fever, malignant tumors, neurological diseases, orthopedic disorders, and many other chronic ailments frequently discover that music helps while away tedious hours and removes flights of the imagination to more pleasant and relaxing spheres of thought. This can be accomplished with the aid of background music, records, books, musical instruments, and singing.

Arrington believes that music is a useful adjunct to physical therapy. Since the active participation of patients whose limbs require remedial exercise is often invaluable, the piano, for example, provides a contraction-relaxation pattern for strengthening the upper extremities that is hardly equaled by any other musical instrument. Many patients with arm casts are able to play an instrument when the casts are properly supported. It has been observed that digital motion in patients with contractures due to burns is frequently much improved by the playing of the piano.

Since almost all children enjoy music that is tuneful, accompanied by simple words, and that relates a story, music is especially applicable as a therapeutic modality for the juvenile audience. It is useful as a vehicle accompanying games, and it also provides a pleasant distraction from the disturbing cries that invade pediatric wards of hospitals. It is of particular value to chronically ill children who are required to stay in bed for long periods of time.

According to Pichot,[2] mentally defective persons are more easily handled when soothing, rhythmic music is performed. It has been found in dentistry that music allays anxiety, raises the pain threshold, and improves the doctor-patient relationship. Many dentists play background musical recordings in their offices during annoying dental procedures; some obstetricians use music to allay anxiety and raise the pain threshold during arduous hours spent by patients in the labor room. Light [3] observed that music in the operating room served as a beneficial adjunct to the administration of local, regional, or spinal anesthesia and that it diminished apprehension in patients. A survey of a group of patients undergoing various major surgical operations indicated a very favorable response to the calming effects of music.

1. Arrington, G. E., Jr.: Music in Medicine, West Virginia M. J. **49**: 152 (June) 1953.
2. Pichot, F.: Effect of Music on Mental Defectives, Ment. Health **9**: 6 (Aug.) 1949.
3. Light, G. A.: Use of Magnetic Recorder, Anesth. & Analg. **28**: 330 (Nov.-Dec.) 1949.

MEDICAL NEWS

ARKANSAS

Dr. Wells Enters Publishing Field.—Dr. Benjamin B. Wells has resigned as head of the department of medicine, University of Arkansas School of Medicine, Little Rock, to become vice-president and senior editor of W. B. Saunders Company, Philadelphia. Dr. Wells, who became associated with the school of medicine in 1942, has served as assistant professor of pathology, medical director of the University Hospital, professor of medicine, and dean of the medical school. He has been professor of experimental medicine and head of the department of medicine at the M. D. Anderson Foundation of the University of Texas Medical Center in Houston and has taught at the Baylor University College of Medicine, Houston. He received the Legion of Merit for services in the European theater during World War II. Dr. Wells is the author of "Clinical Pathology: Application and Interpretation" and "Treatment of Rheumatoid Arthritis: Current Therapy" and is coauthor with Drs. Arthur H. Sanford and J. C. Todd of "Clinical Diagnosis by Laboratory Methods."

CALIFORNIA

Orthopedic Clinic.—St. Francis Memorial Hospital, San Francisco, has opened an orthopedic clinic. Rotating their contributed time, Drs. John F. Cowan, William E. Jensen, Paul W. Klabunde, Alfred M. Palmer, and Richard J. Wagner will be on duty in the clinic, along with the resident physician, every Tuesday or Thursday, 1-2 p. m., in the physical therapy department. Resident physicians will be on orthopedic assignments for three months. The consulting staff of orthopedists consists of Drs. Delbert W. Hand, Leon O. Parker, and Horace C. Pitkin. Appointments for consultation with the resident and clinic staff physicians may be made by calling the physical therapy department (extension 384).

Personal.—Dr. James T. Weston, pathologist of the coroner's office, San Diego, has been given leave of absence for one year. Under a Damon Runyon fellowship awarded by the American Cancer Society, he will study newer methods in cancer detection at the Memorial Center for Cancer and Allied Diseases, New York.——Dr. Alvin G. Foord, director of laboratories at the Collis P. and Howard Huntington Memorial Hospital, Pasadena, for the past 22 years, has resigned but will serve as consulting pathologist.

COLORADO

State Medical Meeting in Denver.—The Colorado State Medical Society will hold its annual meeting at the Shirley-Savoy Hotel, Denver, Sept. 29-Oct. 2 under the presidency of Dr. William A. Liggett, Denver. Presentations by guest speakers include:

Alson E. Braley, Iowa City, The Ophthalmologist an Aid to Diagnosis of General Disease.
Chester B. McVay, Yankton, S. D., Inguinal and Femoral Herniplasty.
Rupert B. Turnbull Jr., Cleveland, Surgical Treatment of Ulcerative Colitis.
Frank C. Combes, New York, Cutaneous Manifestations of Systemic Disease, Part 1.
Howard F. Root, Boston, Treatment of Diabetic Coma and Its Complications.
Mr. Leo E. Brown, Chicago, Medicine's New "Platoon System."
Claude J. Hunt, Kansas City, Mo., Modern Approach to the Treatment of Thyroid Diseases.
Thomas J. Dry, Rochester, Minn., The Gallbladder and Its Relation to Coronary Disease.

Thursday at 9 a. m. a symposium on "Problems Encountered in Congenital Anomalies" will be presented by Drs. Sidney E. Blandford Jr., Robert K. Brown, and James A. Philpott Jr., Denver. Friday, 10:45 a. m., a symposium on "Surgery of Heart Diseases" will be presented by Drs. John B. Grow, Abe Ravin, and R. Parker Allen, Denver. At the annual banquet Friday, 7 p. m., an address on "Freedom of the Press and the Professions" will be delivered by Mr. Palmer Hoyt, editor and publisher of the *Denver Post*.

Physicians are invited to send to this department items of news of general interest, for example, those relating to society activities, new hospitals, education and public health. Programs should be received at least three weeks before the date of meeting.

CONNECTICUT

Discussion on the Tuberculous Patient.—A panel discussion on "Advances in Treatment and Rehabilitation of the Tuberculous Patient Employed in Industry" will be held at Gaylord Farm Sanatorium, Wallingford, Sept. 23, at 3 p. m. A barbecue dinner will be provided at cost by the sanatorium. Interested Connecticut physicians are invited to attend this meeting, which will be sponsored jointly by the Committee on Industrial Health, Connecticut State Medical Society, and the Gaylord Farm Sanatorium.

Dr. Gray Retires from Health Department.—Dr. Albert S. Gray, Hartford, deputy commissioner of the Connecticut State Department of Health, has retired after 25 years of state service. In 1948, Dr. Gray received a citation from the Connecticut Safety Society for his work in the prevention of occupational illness in the state. He was the first chairman of the industrial hygiene section of the American Public Health Association and is a past president of the American Conference of Governmental Industrial Hygienists.

GEORGIA

County Health Board Establishes Advisory Council.—The Richmond County board of health recently appointed a special medical advisory committee consisting of the following Augusta physicians: Virgil P. Sydenstricker, Harry B. O'Rear, Walter L. Shepeard, and William S. Boyd.

Dr. Elkin to Retire from Faculty.—Dr. Daniel C. Elkin, chairman, department of surgery, Emory University School of Medicine, will retire from the faculty at the end of the 1953-1954 academic year but will maintain an advisery and consulting relationship with the school where since 1939 he has occupied the Joseph B. Whitehead chair of surgery. Dr. Elkin, a nephew of the late Dr. W. S. Elkin, dean of the Atlanta Medical College at the time it became the medical school of Emory University and first dean of the new school, served as surgeon-in-chief of the Grady Memorial Hospital in Atlanta from 1939 to 1948. He is now consultant in surgery to the Surgeon General, chief civilian consultant in surgery for the Third Army, and consultant to the Walter Reed Army Medical Center, Washington, D. C. Dr. Elkin has served as president of the American Surgical Association, the Society for Clinical Surgery, and the Southern Surgical Association, and special consultant to the U. S. Public Health Service (National Institutes of Health).

IDAHO

Society News.—The Southwest Idaho District Medical Society has elected Dr. Lowell B. Privett, Boise, president; Dr. Lester L. Shupe, Caldwell, vice-president; Dr. Max F. Bell, Boise, secretary; and Dr. A. Curtis Jones, Boise, treasurer.——At the annual meeting of the Idaho Chapter of the American College of Surgeons in Boise Dr. Arthur V. Jones Sr., Boise, was elected president, Dr. Albert B. Pappenhagen, Orofino, vice-president, Dr. James H. Hawley, Boise, secretary, and Dr. Charles B. Beymer, Twin Falls, counselor.

ILLINOIS

Conference on Exceptional Children.—The Illinois Commission for Handicapped Children will sponsor the 10th Governor's Conference on Exceptional Children Sept. 25 at the Palmer House, Chicago. Group meetings will be arranged for the handicapped child in preschool, in the regular school

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program, in the community recreation program, and in institutions. During a symposium on "Generic Casework and the Handicapped Child," Dr. M. Jeanne Spurlock, psychiatrist, Institute for Juvenile Research, Chicago, will discuss "Attitudes of Caseworkers That Help or Hinder." Featured speaker for the conference luncheon will be the Rev. Harold Wilke, pastor of St. Paul's Evangelical and Reformed Church, Crystal Lake, Ill., former chaplain in military hospitals, and for several years a faculty member of the Menninger Foundation school of psychiatry in Topeka, Kan. "A Reporter's Round-Up of the Conference" will be presented by Mr. Eugene J. Taylor, staff writer, *New York Times*, and co-author with Dr. Howard A. Rusk, New York, of "New Hope for the Handicapped."

Chicago

Society News.—The north side branch of the Chicago Medical Society will meet at 8 p. m., Oct. 1, in the ballroom of the Drake Hotel, Lake Shore Drive and Upper Michigan Ave. Dr. Hiram T. Langston, Riverside, Ill., will speak on "Carcinoma of the Lung."

Personal.—Dr. Ben W. Lichtenstein presented a paper on "Sturge-Weber-Dimitri Syndrome Cephalic Form of Neurocutaneous Hemangiomatosis" at the International Neurological Congress in Lisbon, Portugal. Dr. Percival Bailey, who also attended that congress and the German Neurological Congress at Munich, has been invited to address a meeting at Seville, Spain, on "Psychomotor Epilepsy," and Dr. Paul C. Bucy will present "Experimental Studies on the Temporal Lobe." All are professors at the University of Illinois College of Medicine.

IOWA

Dr. Heeren Named Deputy Health Commissioner.—Dr. Ralph H. Heeren, Des Moines, has been named Iowa state deputy commissioner of health. He formerly served as associate professor of preventive medicine, State University of Iowa College of Medicine, Iowa City. Affiliated with the Iowa state department of health since 1948, he was previously director of the division of communicable diseases, state health department of Oklahoma. He has served on the faculties of Western Reserve University School of Medicine, Cleveland, and Tulane University of Louisiana School of Medicine, New Orleans; has been affiliated with Mt. Sinai Hospital, Cleveland, and Albany (N. Y.) Hospital; and was a Rockefeller Foundation fellow in the school of public health at Harvard Medical School, Boston.

Personal.—Dr. Paul D. Anneberg, Carroll, has been made president of the Rotary Club.——Dr. J. Frank Aldrich, Clarinda, who recently retired from active medical practice after 55 years, was honored during an open house program at Hand Memorial Hospital in Shenandoah. During the ceremonies Dr. Aldrich presented the hospital board with a check for $1,000 in memory of his wife.——In recognition of his 50 years of practice Dr. Henry D. Jones was guest of honor at a meeting of the Schleswig business men's club, which presented him with a testimonial scroll. The scroll listed his many services to the community, including "hardships of his early practice, during the horse and buggy days, and before the era of good roads; willingness during his professional career to answer every call, without thought of personal comfort or financial remuneration; and genuine interest in community welfare."——Dr. Edmund G. Zimmerer, Des Moines, former director of the Division of Cancer Control, Iowa State Health Department, and member of the National Cancer Institute Cancer Control Committee, has been appointed Iowa State commissioner of health for a four year term.——Dr. Louis H. Kornder, Davenport, is retiring after 35 years of practice and will devote himself to civic interests. Dr. Kornder is president of the board of the Davenport museum, vice-president of the Municipal Art Gallery, curator of the Iowa State Historical Society, and a trustee of Dubuque University. ——Dr. Eugene Tinsman, who has practiced in Orient for 50 years, was recently honored by an open house in the school building. Dr. Tinsman has served 12 years on the Orient school board.

KANSAS

Cancer Diagnostic Clinic at Chanute.—Neosho Memorial Hospital, Chanute, recently organized a cancer diagnostic clinic, after having established a cancer registry and follow-up service a year ago. Cancer control activities in Kansas are jointly sponsored by the Kansas Medical Society's committee on cancer control, the Kansas Division of the American Cancer Society, and the cancer section of the Kansas State Board of Health.

KENTUCKY

State Medical Meeting in Louisville.—The annual meeting of the Kentucky State Medical Association will convene at the Columbia Auditorium, Louisville, Sept. 22-24. The general session, which is designated as the Lewis Rogers Memorial meeting, will be called to order by the president, Dr. Guthrie Y. Graves, Bowling Green, at 9 a. m. Tuesday. At the president's luncheon, Roof Garden, Brown Hotel, at noon, Dr. Edward J. McCormick, Toledo, Ohio, President of the American Medical Association, will speak on "Firm Ground for Medicine." Other guest speakers include:

George T. Harrell, Winston-Salem, N. C., How Antibiotics Work.
Robert M. Zollinger, Columbus, Ohio, Indications for Surgery in the Jaundiced Patient.
Orville B. Coomer, D.D.S., Louisville, Growing Interdependence of Medicine and Dentistry.
Harold E. Harris, Cleveland, Indications and Contraindications of Tonsillectomy.
Paul H. Holinger, Chicago, Bronchial Obstruction: Its Significance in Pulmonary Disease.
Mr. Leo E. Brown, Chicago, PR Profits.
Col. William H. Amspacher, Fort Sam Houston, Texas, Early Care of the Severe Burn.
Donald A. Covalt, New York, Rehabilitation of the Hemiplegic Patient.
Mr. C. Joseph Stetler, Chicago, The Legislative Program and Activities of the A. M. A.
H. Close Hesseltine, Chicago, Changing Patterns in Obstetric Practice.

The presidential address will be delivered Wednesday, 10 a. m., and the oration in medicine, "Heart Disease From the Patient's Viewpoint," will be given at 11:30 a. m. by Dr. Paul W. Simpson, Covington. The annual banquet will be held Thursday 7:30 p. m. in the Crystal Ballroom of the Brown Hotel (dress optional). Mr. Allan Kline, president, American Farm Bureau, will speak on "Promises and Prospects." The woman's auxiliary will meet concurrently at the Brown Hotel where, on Wednesday, Dr. Graves and Dr. J. Duffy Hancock, Louisville, president-elect of the Kentucky State Medical Association, will participate in the panel on public relations.

MICHIGAN

Meeting of Medical Assistants.—The fourth annual convention of the Michigan State Medical Assistants Society will be held at the Pantlind Hotel, Grand Rapids, Sept. 23-24. On Wednesday Dr. Luther C. Carpenter Jr., Grand Rapids, will speak on "Cancer," and Dr. David G. Dickinson, Ann Arbor, will discuss "Poliomyelitis." Luncheon at 12:30 p. m. will follow a presentation on "Legal Aspects in Medicine" by William A. Perkins, LL.B., Grand Rapids. At the banquet, 7:30 p. m., A. E. Schneider, Ph.D., Kalamazoo, will have as his topic "Your Attitude and You." Thursday morning Dr. Edwin O. Pearson, Kalamazoo, will discuss "X-Ray and X-Ray Therapy," and Dr. Anthony F. Stiller, Oshtemo, will talk on "Tuberculosis." The president's luncheon, 12:30 p. m., will be followed by a presentation on "History of Medicine" by Harvey M. Merker, D.Sc., consultant to Parke, Davis & Company, Detroit.

State Medical Meeting in Grand Rapids.—The Michigan State Medical Society will hold its annual session at the Pantlind Hotel, Grand Rapids, Sept. 23-25 under the presidency of Dr. Reader J. Hubbell, Kalamazoo. Discussion conferences will be conducted each day from 12 to 1 p. m. There will be a public meeting Wednesday, 8:30 p. m., in the Civic Auditorium, where many of the scientific sessions will convene. After the presidential address and induction of members into the 50 year club, Dr. Edward J. McCormick, Toledo, Ohio, President of the American Medical Association, will speak on

"Organized Medicine—Its Progress and Problems." At 9:15 p. m. the Andrew P. Biddle lecture on "World Affairs and Foreign Policy" will be delivered by U. S. Senator Homer Ferguson, Detroit. On Thursday, which has been designated as general practice day, the following program will be presented by out-of-state speakers:

Chevalier L. Jackson, Philadelphia, What Can Bronchoscopy Do for the General Practitioner?
Michael L. Mason, Chicago, The Crushed Hand.
George E. Shambaugh Jr., Chicago, Management of Hearing Impairments.
Blair Holcomb, Portland, Ore., Patients Are People—A Physician's Soliloquy.
Allan C. Barnes, Cleveland, Management of the Infertile Couple.
Arthur T. Hertig, Boston, Genesis of Uterine Cancer, Cervical and Endometrial.
Victor A. Najjar, Baltimore, Jaundice in Infancy and Childhood.
Louis A. M. Krause, Baltimore, Physical Examination.

The following presentations will be made by other out-of-state speakers during the sessions:

Walter G. Maddock, Chicago, Development and Management of Gastrointestinal Distention.
William D. Holden, Cleveland, Place of Radioactive Isotopes in Clinical Practice.
Everett I. Evans, Richmond, Va., Treatment of the Severely Burned.
Frank Hinman Jr., San Francisco, Clinical Use of Testosterone.
Leo M. Taran, Roslyn, L. I., N. Y., Management of Rheumatic Fever and Rheumatic Heart Disease.
William L. Benedict, Rochester, Minn., Multiple Sclerosis.
David P. Findley, Omaha, The Postcesarean Baby.
O. Spurgeon English, Philadelphia, Treatment of Psychosomatic Illness.
Jacob A. Bargen, Rochester, Minn., Problems of Nutrition in Ulcerative Disease of the Digestive Tract (Beaumont lecture).
John G. Downing, Boston, Oral and Cutaneous Clues in Medicine and Surgery.
Leo G. Rigler, Minneapolis, Hepatography—The Roentgen Examination of the Liver.
John W. Knutson, D.D.S., Washington, D. C., An Evaluation of the Grand Rapids Water Fluoridation Supply.
Paul R. Cannon, Chicago, Successes and Failures in Relation to the Problem of Parenteral Alimentation.
Harold J. Magnuson, Chapel Hill, N. C., Interpretation of Positive Serologic Tests for Syphilis in Clinically Negative Patients.
Norman M. Keith, Rochester, Minn., Factors in Treatment of Edema.
Leslie N. Gay, Baltimore, The Dramatic Story of Dramamine.
Julian Johnson, Philadelphia, Present Status of the Surgery of Mitral and Aortic Stenosis.

MINNESOTA

Dr. Melby Night.—Dr. Benedik Melby, Blooming Prairie, was honored by the community on "Dr. Melby Night," when he was presented with a gold clock and pen desk set and a framed, hand-lettered scroll commemorating his 50th anniversary in the practice of medicine. Dr. Melby has been a member of the board of education for 39 years. In February, 1951, he was awarded a scroll by the Minnesota Educational Association for his interest and work in the public schools.

NEW YORK

Lecture on Psychosomatic Gynecology.—Dr. William S. Kroger, Chicago, will speak on "Psychosomatic Gynecology," Sept. 24, 8:30 p. m. in the Jewish Sanitarium and Hospital for Chronic Diseases, 86 E. 49th St., Brooklyn. The lecture is presented by the gynecologic service.

Personal.—After 14 years of service at the Ray Brook State Tuberculosis Hospital in Ray Brook, Dr. Joseph Gordon, principal thoracic surgeon, has resigned and has entered the private practice of thoracic surgery at 106 S. Girard St., Albuquerque, N. Mex.

NORTH CAROLINA

University News.—Dr. David Cayer, who has been associate professor of internal medicine, Bowman Gray School of Medicine of Wake Forest College, Winston-Salem, since July 1, 1949, has been promoted to professor of gastroenterology.

Personal.—Dr. W. Nelson Thompson, Raleigh, has been appointed medical director of the Durham Life Insurance Company.——Dr. Coy C. Carpenter, dean of the Bowman Gray School of Medicine, Winston-Salem, has been awarded a Fulbright visiting professorship in pathology for a nine month lecture series in Cairo, Egypt.——Joseph E. Markee, Ph.D., professor of anatomy, Duke University School of Medicine, Durham, has been chosen as the fourth honorary member named by the American Orthopedic Association since its founding in 1887. The society honored Dr. Markee for his work in audiovisual education, particularly development of teaching aids in orthopedic surgery. Dr. Markee was made an honorary member of the American Academy of Orthopedic Surgery in 1950.——Dr. Wingate M. Johnson, Winston-Salem, served as moderator for a panel on "Aging" at the annual meeting of the North Carolina Public Health Association in Nags Head, Sept. 10-12.

PENNSYLVANIA

Anesthesiologists Meet in Pittsburgh.—The annual meeting of the Pennsylvania Society of Anesthesiologists will be held in Pittsburgh Sept. 25-26. Out-of-state speakers include:

C. Paul Boyan, New York, Ganglionic Blockade During Anesthesia.
Emery A. Rovenstine, New York, Cyclopropane.
Anthony J. Bayuk, Youngstown, Ohio, Use of Orthoxine in Anesthesia.
Maurice S. Segal, Boston, Advances in Inhalation Therapy.

Personal.—For his entry "Physicians and Medicine on Stamps," Dr. Herbert B. Shearer, Worcester, received the grand award at the International Stamp Exhibition held at the National Philatelic Museum, Philadelphia, under the auspices of the American Topical Association. Dr. Shearer's collection of "U. S. Christmas Seals and Red Cross Stamps of the World" won first prize in the section of "International Organizations."——Dr. Herman W. Rannels, chief of obstetrics and gynecology, St. Joseph's Hospital, Lancaster, has become one of the directors of the Hunterdon Medical Center in Flemington, N. J. The directorship includes a teaching position on the staff of New York University-Bellevue Medical Center, with which the center is affiliated.

Philadelphia

Phi Delta Epsilon Lecture.—Beta Zeta chapter, Phi Delta Epsilon fraternity, Hahnemann Medical College and Hospital of Philadelphia, has announced its annual lectureship to the medical profession, Oct. 2, 8 p. m. at Klahr Auditorium, 235 N. 15th St. Dr. Nicholson J. Eastman, Baltimore, will speak on "25 Years of Obstetric Pain Relief: What Have We Learned?"

Mental Health Director Wanted.—The Philadelphia Department of Public Health has an opening for a director of its new division of mental health. Applicants are required to have seven years of clinical psychiatric experience, including three years in charge of other psychiatrists, one year in children's psychiatry, and three years in approved training centers, that have led to certification by the American Board of Psychiatry and Neurology. The starting salary is $13,442. Closing date for applications is midnight, Oct. 21.

TEXAS

General Practitioners Meet in Dallas.—The annual scientific assembly of the Texas Academy of General Practice will be held at the State Fair Auditorium in Dallas, Sept. 21-23. Out-of-state guest speakers include:

Francis L. Chamberlain, San Francisco, Specific Treatment of Hypertension. Recent Advances in Electrocardiography of Importance in General Practice.
Henry H. Turner, Oklahoma City, Endocrine Disorders of Childhood. Psychosomatics and Pseudoendocrinopathies.
Edgar Hull, New Orleans, Hints in Recognition of Certain Endocrine Diseases. Asymptomatic Organic Disease.
Henry W. Brosin, Pittsburgh, Interviewing Techniques. Depressive Reactions in General Practice.
Somers H. Sturgis, Boston, Problems of Sterility. Diagnosis and Treatment of Amenorrhea.
Benjamin J. Wolpaw, Cleveland, Retinal Changes in Hypertensive Vascular Disease. External Diseases of the Eye as Seen by the General Practitioner.
Marcus J. Stewart, Memphis, Diagnosis and Treatment of Minor Foot Problems. Differential Diagnosis of Low Back Pain.
Alton Ochsner, New Orleans, Diagnosis of Cancer of the Lung. Diagnosis and Treatment of the Post-Phlebitic Extremity.
Philip Thorek, Chicago, Appendicitis. Diagnosis and Treatment of Breast Lesions.
Henry M. Weyrauch Jr., San Francisco, Pitfalls in Urologic Diagnosis. Office Treatment of Common Urologic Conditions.

The fee is $20. Nonmembers are invited to attend. Entertainment for the physicians' wives has been arranged.

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Industrial Health Conference in Houston.—The Houston Chamber of Commerce will sponsor the sixth annual Gulf Coast Conference on Industrial Health at the Shamrock Hotel, Houston, Oct. 1-3. During the symposium on "Employment of the Cardiac" Thursday afternoon, Dr. Harold M. Marvin, New Haven, Conn., will discuss "The Physician's Dilemma," and Dr. Kenneth E. Markuson, Helena, Mont., will speak on "The Insurance Carrier Views the Problem." Vice Admiral Ross T. McIntire (MC), U. S. N., Ret., Washington, D. C., will speak. Dr. Robert L. King, Seattle, will serve as moderator for a panel discussion at 4:30 p. m. during which previous speakers will participate in a question and answer period. Other physicians participating in the Thursday program include Dr. Theodore M. Frank, Texas City, who will serve as co-chairman of the morning session; Dr. Paul V. Ledbetter, Houston, co-chairman of the luncheon session; Drs. Sidney Schnur and Valliant C. Baird, Houston, chairman and co-chairman, respectively, of the afternoon session. The morning session will be presented by Dr. George M. Saunders, New York ("The Executives' Health Problem"), Dr. Charles H. Hine, Berkeley, Calif. ("Methods of Establishing Safe Handling Procedures for Compounds Developed by the Petro-Chemical Industry"), and Dr. Robert E. Eckardt, Linden, N. J. ("A Medical Research Program in Industry"). Speakers for the luncheon sessions are Dr. Robert L. King, Seattle, who will report on the American Heart Association, of which he is president, and Dr. Marvin, New Haven, Conn., who will discuss "The Executive and His Heart." At the evening session in the Prudential Building Auditorium, Vice Admiral McIntire will present "Recovery of Human Resources Through Rehabilitation." Friday morning will be devoted to a symposium on "The Noise Problem in Industry":

C. Richard Walmer, Pittsburgh, Noise in Industry—The General Problem.
James H. Sterner, Rochester, N. Y., Industrial Noise—Its Medical Aspect.
Rear Admiral Herbert Lamont Pugh, Washington, D. C., Noise, Noxious or Nice.
Jack L. Bangs, Ph.D., Houston, Testing for Hearing Loss and Its Application to Industry.

Friday afternoon Dr. Arthur J. Vorwald, Saranac Lake, N. Y., will present "Medical Aspects of Air Pollution" in a symposium on "Waste Disposal Problems," which will be preceded by a film showing on "The City That Disappears."

GENERAL

United Cerebral Palsy Appoints Chairman.—Dr. H. Houston Merritt, director of the Neurological Institute of the Columbia-Presbyterian Medical Center, New York, has been appointed chairman of the board, which coordinates the work of the organization's five volunteer medical and professional boards and sets over-all policy. Dr. Merritt succeeds Dr. Sidney Farber, professor of pathology at Harvard Medical School, Boston, who will continue to serve as chairman of the Research Advisory Board. Taking over as chairman of the Clinical Advisory Board, a position formerly held by Dr. Merritt, is Dr. Charles F. McKhann, professor of pediatrics at Jefferson Medical College, Philadelphia.

Cancer Society Fellowships.—The American Cancer Society has announced that its program of clinical fellowships will continue through the next institutional training year to June 30, 1955. The program for the coming year will include support for clinical traineeships. These traineeships, which are opportunities for training for physicians at a level somewhat lower than the clinical fellowship, carry stipends up to a maximum of $3,600. Fellowships and traineeships will be made available primarily to teaching institutions approved by the Council on Medical Education and Hospitals of the American Medical Association. Applications should be addressed to Dr. Brewster S. Miller, American Cancer Society, 47 Beaver St., New York 4. The deadline for filing applications is Oct. 5. Application forms are not necessary.

Medical Illustrators Meet in Baltimore.—The annual convention of the Association of Medical Illustrators will be held at the Johns Hopkins Medical Institutions, Baltimore, Oct. 5-7. Participating in the program will be Drs. Alan C. Woods, Baltimore ("The Value of Art in Ophthalmology as Opposed to Photography"), Victor A. McKusick, Baltimore ("The Hutchinson Collection"), and David S. Ruhe, Chicago ("Role of the Illustrator in a Changing Medical Audiovisual Program in the Medical Center"). Dr. Jonas S. Friedenwald, Baltimore, associate professor of ophthalmology at Johns Hopkins Medical School since 1931, will deliver the Brödel Memorial lecture on "Advances in Visual Physiology and the Portrayal of Depth of Space in Painting" after the dinner meeting on Tuesday. Monday at 8 p. m. there will be a tour of the new photographic department, Johns Hopkins Hospital Reconstruction Unit #1. At 8:45 p. m. movies will be shown on "Embryology of the Eye," "Amebic Dysentery," and "Carcinoma of the Prostate."

Meeting of Neurosurgical Society.—The Neurosurgical Society of America will hold its annual meeting at the Broadmoor Hotel in Colorado Springs, Colo., Sept. 23-26 under the presidency of Dr. William F. Meacham, Nashville, Tenn. Thursday morning will be devoted to a "Forum on Clinical Problems as Related to Diagnosis," for which the moderators will be Drs. Francis J. Otenasek, Baltimore, Theodore B. Rasmussen, Chicago, and Christian Keedy, Miami, Fla. Dr. Kenneth E. Livingston, Portland, Ore., will serve as chairman for the Thursday afternoon symposium on "Present Status of Psychosurgery Including the Legal Aspects." Participants will be Drs. C. Douglas Hawkes, Memphis, Tenn., Alfred Uihlein, Rochester, Minn., Arthur A. Morris, Washington, D. C., and John R. Green, Phoenix, Ariz. There will be a panel Friday morning on "Metabolic and Electrolytic Problems in Neurosurgery," in which Drs. Eben Alexander Jr., Winston-Salem, N. C., Irving Cooper, New York, Jean M. Hawkes, Memphis, Tenn., and Robert M. Salassa, Rochester, Minn., will participate. The golf tournament will be on Friday afternoon. The banquet (formal), 7:30 p. m., will be preceded by a cocktail hour. Speaking by invitation Saturday morning will be Drs. Arthur A. Morris and Clemmer M. Peck, Washington, D. C. ("Anatomy of Anterior Cerebral Artery as Demonstrated by Arteriography") and Drs. Lester A. Mount and Juan M. Taveras, New York ("Study of Collateral Circulation of the Brain Following Various Ligations Intracranially and in the Neck"). At 6 p. m. Saturday there will be a steak fry in the canyon.

Pediatricians Meet in Miami.—The American Academy of Pediatrics will hold its annual meeting in the Municipal Auditorium, Miami, Fla., Oct. 6-9 under the presidency of Dr. Philip S. Barba, Philadelphia. Two day instruction courses (enrollment limited to from 35 to 50 in each seminar) will be presented Oct. 4-5. Fee for each seminar is $30. The following presentations will be made:

Lawson Wilkins, Baltimore, Pediatric Endocrinology.
Helen B. Taussig, Baltimore, Heart Disease in Children.
Randolph K. Byers, Boston, Pediatric Neurology.
Richard E. Wolf, Cincinnati, Pediatric Psychiatry.
Julius B. Richmond, Chicago, Pediatric Psychiatry.
Susan C. Dees, Durham, N. C., Pediatric Allergy.
Weston M. Kelsey, Winston-Salem, N. C., Electrolyte and Mineral Balance.
Harry H. Gordon, Baltimore, Problems in the Management of the Premature and Newborn Infant.
J. Cyril Peterson, Nashville, Tenn., Pediatric Hematology.

Round-table discussions (half day courses with limited enrollment for from 35 to 50) will be run concurrently with the general sessions Oct. 6-8. Fee for each discussion is $5. During the general sessions there will be symposiums on "Health Evaluation of the Normal Child" (chairman, Dr. Thomas E. Shaffer, Columbus, Ohio); "Physiology of the Gastrointestinal Tract and Its Functional Disturbances" (chairman, Dr. L. Emmett Holt Jr., New York); "Respiratory Problems Affecting the Premature and Full-Term Infant" (chairman, Dr. Stewart H. Clifford, Boston); and "Respiratory Viral Diseases" (chairman, Dr. Joseph Stokes Jr., Philadelphia). Dr. Sidney Farber,

Boston, will conduct clinical pathological conferences Monday, 2-5 p. m. and with Dr. Waldo E. Nelson, Philadelphia, Friday, 11:20 a. m. Thursday morning the Borden award paper will be read by Dr. Lawson Wilkins, Baltimore; the first Mead Johnson award paper by Drs. Frederick C. Robbins and Thomas H. Weller, Boston; and the second Mead Johnson award paper by Dr. Margaret H. Smith, New Orleans. The annual banquet will be held Thursday evening.

Southwestern Surgical Congress.—This congress will hold its annual meeting at the Hotel Utah, Salt Lake City, Sept. 21-23 under the presidency of Dr. Louis P. Good, Texarkana, Ark. Morning sessions will open at 8 a. m. with the showing of motion pictures. Monday, 11 a. m., Dr. Warren H. Cole, Chicago, will present "Management of Strictures of the Common Duct." At 9:50 a. m. Drs. James Barrett Brown, St. Louis, and Minot P. Fryer, St. Louis, will speak on "Management of Hemangioma and Nevi." At 2 p. m. Dr. William H. Moretz, Salt Lake City, will serve as moderator for a panel on trauma, with the following participants, all of Salt Lake City: Drs. Woodrow Nelson, Chester B. Powell, Paul A. Pemberton, Preston R. Cutler, and Robert R. Robinson Jr. At 3:30 p. m. the sessions will adjourn for a trip to Brighton Resort. Tuesday, 9:20 a. m., Dr. Henry Plenk, Salt Lake City, will speak by invitation on "Gastric Ulcers—A Correlative Study," after which Dr. Samuel F. Marshall, Boston, will discuss "The Problem of Jejunal Ulcer." At 11 a. m. Dr. Everett D. Sugarbaker, Jefferson City, Mo., will present "Internal Mammary Node Removal in Continuity with Radical Mastectomy." At 2 p. m. Dr. Marshall will be guest moderator for a panel on "Management of Goitre," in which Drs. Lawrence P. Engel, Kansas City, Mo., Geno Saccomanno, Grand Junction, Colo., and John Z. Bowers, Salt Lake City, will serve as participants. At 3 p. m. Dr. Goode will deliver the presidential address, "The Methods and Manners of Great Men of Medicine." Cocktails, 6:30-7:45 p. m., at the Alta Club will precede the dinner dance. Wednesday, 9:40 a. m., Dr. Philip B. Price, Salt Lake City, will speak on "An Experimental Study of Burn Wounds." At 11 a. m. Dr. Marshall will discuss "Surgical Management of Regional Enteritis and Its Complications." At 2 p. m. there will be a panel on "Diseases of the Colon and Rectum" with Dr. Thomas G. Orr, Kansas City, Kan., as moderator, and Drs. Edgar J. Poth, Galveston, Texas, Kenneth C. Sawyer, Denver, and Leo J. Starry, Oklahoma City, as participants. Luncheon and round-table discussions will be held daily, 12:30-2 p. m.

College of Surgeons Meeting in Chicago.—The annual clinical congress of the American College of Surgeons will be held at the Conrad Hilton, Chicago, Oct. 3-9. Dr. Harold L. Foss, Danville, Pa., president, will preside at the opening evening session, at which Dr. Fred W. Rankin, Lexington, Ky., will be installed as president for 1954. Subjects for panel discussion include "Management of Gastric Ulcers," "Carcinoma of the Mouth," "Blood Transfusions: Complications and Their Management," "Polyposis of the Colon," "Surgery of the Spleen," and "Malignant Thyroid Disease." Panel discussions in the surgical specialties will be held Friday afternoon. On Tuesday, 2-5 p. m., there will be a symposium on trauma at the Eighth Street Theatre. Wednesday, 2-5 p. m., a symposium on cancer will be held at the John B. Murphy Memorial Auditorium. Thursday, 1:30-3 p. m., a symposium on graduate training in surgery and the surgical specialties is scheduled for the Conrad Hilton. A motion picture symposium on unusual conditions encountered in abdominal surgery will be held Thursday, 8-10 p. m. Clinical pathological conferences are scheduled for 1:30 p. m. Monday and Wednesday. Surgical procedures at Albert Merritt Billings Hospital will be color televised to the hotel every day under the sponsorship of Smith, Kline and French Laboratories of Philadelphia. Twelve forums on fundamental surgical problems will present the latest research findings. At an evening session Monday, Dr. Foss will deliver the presidential address. The eighth Martin Memorial lecture will be delivered by Sir James Paterson Ross, vice-president, Royal College of Surgeons of England, who will discuss "Science and Surgery." During the Wednesday evening session Dr. J. William Hinton, New York, will speak on "Cardiac Arrest: Etiology and Management from a Study of Over 1,300 Cases," and Dr. Francis D. Moore, Boston, will discuss "Metabolic and Electrolyte Abnormalities in Severely Ill Cardiac Patients and Individuals with Advanced Cancer." "Present Day Problem of Nonpenetrating Abdominal Trauma" is the subject of the Oration on Trauma to be delivered by Dr. William L. Estes Jr., Bethlehem, Pa. The Friday evening session at Medinah Temple will close with an address on "The Responsibility of a Heritage of Idealism" by Dr. Fred W. Rankin. Registration fee for nonfellows and fellows whose dues are not paid to Dec. 31, 1952, will be $20. Registration forms may be obtained from the American College of Surgeons, 40 E. Erie St., Chicago 11.

Prevalence of Poliomyelitis.—According to the National Office of Vital Statistics, the following number of reported cases of poliomyelitis occurred in the United States, its territories, and possessions in the weeks ended as indicated:

Area	Sept. 5, 1953: Paralytic Type	Sept. 5, 1953: Total Cases Reported	Sept. 6, 1952
New England States			
Maine	10	22	10
New Hampshire	..	1	5
Vermont	3	7	..
Massachusetts	17	40	40
Rhode Island	6	22	7
Connecticut	5	32	32
Middle Atlantic States			
New York	58	196	154
New Jersey	40	68	61
Pennsylvania	..	78	130
East North Central States			
Ohio	27	178	156
Indiana	..	39	112
Illinois	43	165	295
Michigan	47	159	275
Wisconsin	..	56	198
West North Central States			
Minnesota	45	188	283
Iowa	2	30	241
Missouri	32	85	139
North Dakota	1	14	20
South Dakota	..	8	39
Nebraska	4	9	211
Kansas	..	35	162
South Atlantic States			
Delaware	..	1	12
Maryland	19	39	7
District of Columbia	1	5	19
Virginia	11	22	58
West Virginia	12	31	54
North Carolina	12	30	34
South Carolina	4	8	16
Georgia	6	15	28
Florida	9	20	30
East South Central States			
Kentucky	11	20	124
Tennessee	9	25	55
Alabama	4	8	23
Mississippi	1	8	21
West South Central States			
Arkansas	12	17	25
Louisiana	16	24	12
Oklahoma	2	22	95
Texas	19	62	196
Mountain States			
Montana	5	19	16
Idaho	..	2	11
Wyoming	..	4	3
Colorado	6	8	48
New Mexico	..	5	29
Arizona	7	29	14
Utah	..	19	4
Nevada	..	..	2
Pacific States			
Washington	3	30	102
Oregon	16	26	32
California	89	183	166
Territories and Possessions			
Alaska	1	..	..
Hawaii	1	..	3
Puerto Rico	..	..	..
Total	616	2,132	3,820

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PHILIPPINE ISLANDS

Pilot Project in Bilharziasis Control.—Plans have been drawn for construction of a technical center on the Island of Leyte to serve as the headquarters for a six year pilot project in bilharziasis control. This building will house research laboratories and administrative offices, as well as an aquarium for the study of snails, vectors of bilharziasis. It is estimated that 40 persons will be employed at the center. A three man international team to be provided by WHO for this project will work with a matching team of experts from the Department of Health of the Philippines. The aim of the pilot project will be to determine the best method of controlling bilharziasis, which is believed to afflict hundreds of thousands of persons in the Philippines. The length of the project is necessitated by the fact that there is insufficient knowledge concerning practicable and economical means in the control of this disease.

CANADA

National Immunization Week.—National Immunization Week will be observed from Sept. 27 to Oct. 3, under the leadership of the Health League of Canada. This is a nationwide educational campaign to bring to the attention of all parents the protection against diphtheria, whooping cough, lockjaw, and smallpox that is available for their children. Dr. Franklyn O. Wishart, professor of hygiene and preventive medicine, University of Toronto, is chairman of the Health League's National Immunization Committee.

FOREIGN

Aero Medical Meeting.—The interim meeting of the Aero Medical Association will be held under the auspices of the French Speaking Branch at the University of Brussels Medical School, Brussels, Belgium, Sept. 24-27. Rear Admiral Bertrand Groesbeck Jr. (MC) USN (Ret.), Washington, D. C., is president of the association, and Major General Charles Sillevaerts, former chief of the Belgian Air Force Medical Service, is president of the French speaking group. A symposium on jet pilot fatigue will be held Thursday. Other topics to be discussed include: "Modifications of Arterial Pressure of Men During Breathing in Overpressure," "Threshold of Organic Disturbances During Inhalation of Pure Oxygen," "Some Changes of Performances with Age and Their Reference to Aircrew," "Medical Aspects of Abnormal Use of Ejectable Seats," "Research on Human Factor in Accidents," "Is Electrophrenic Respiration of Any Use in the Reanimation of Subjects in Anoxemic Fainting State?" and "Research on the Pharmaco-Dynamic Factors Which Can Modify Endurance to Anoxia."

DEATHS IN OTHER COUNTRIES

Dr. Erasto Gaertner, mayor of the city of Curitiba, State of Parana, Brazil, died May 19, 1953. Dr. Gaertner was professor of the surgical clinic, ex-professor cathedrat of obstetrics, and professor of the urologic clinic of the faculty of the University of Parana. He was founder and director of the Institute of Medicine and Surgery of Parana, as well as founder of the clinic of tumors of the Institute of Medicine and Surgery of Parana. He served as deputy and constituent of the State Assembly of Parana in 1935, as deputy constituent of the National Assembly in 1946, and he was a member of the committee on education and culture of the Deputy Chambers.

CORRECTION

Carbon Dioxide Inhalation in Muscular Rigidity.—In the article on this subject in THE JOURNAL, Aug. 22, the word alkylosis in the second paragraph on page 1624 under the heading "Background" should be alkalosis.

MEETINGS

AMERICAN MEDICAL ASSOCIATION: Dr. George F. Lull, 535 North Dearborn St., Chicago 10, Secretary.
1953 Clinical Meeting, St. Louis, Dec. 1-4.
1954 Annual Meeting, San Francisco, June 21-25.
1954 Clinical Meeting, Miami, Florida, Nov. 30-Dec. 3.
1955 Annual Meeting, Atlantic City, N. J., June 6-10.
1955 Clinical Meeting, Boston, Nov. 29-Dec. 2.

NATIONAL CONFERENCE ON PHYSICIANS AND SCHOOLS, Moraine-on-the-Lake Hotel, Highland Park, Ill., Sept. 30-Oct. 2. Dr. W. W. Bauer, 535 N. Dearborn St., Chicago 10, Director.

AMERICAN ACADEMY FOR CEREBRAL PALSY, Western Hills Hotel, Fort Worth, Texas, Oct. 30-31. Dr. Harry E. Barnett, 116 S. Michigan Blvd., Chicago 3, Secretary.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, Palmer House, Chicago, Oct. 12-16. Dr. W. L. Benedict, 100 First Avenue Bldg., Rochester, Minn., Executive Secretary.

AMERICAN ACADEMY OF PEDIATRICS, Municipal Auditorium, Miami, Fla., Oct. 6-9. Dr. E. H. Christopherson, 610 Church St., Evanston, Ill., Secretary.

AMERICAN ACADEMY OF TROPICAL MEDICINE, Brown Hotel, Louisville, Ky., Nov. 12-14. Dr. E. Harold Hinman, University of Puerto Rico, School of Medicine, San Juan 22, P. R., Secretary.

AMERICAN ASSOCIATION OF BLOOD BANKS, LaSalle Hotel, Chicago, Oct. 17-20. Miss Marjorie Saunders, 3500 Gaston Ave., Dallas 4, Texas, Secretary.

AMERICAN ASSOCIATION OF MEDICAL CLINICS, Congress Hotel, Chicago, Oct. 9-11. Dr. Edwin P. Jordan, P. O. Box 114, Charlottesville, Va., Executive Director.

AMERICAN ASSOCIATION OF MEDICAL RECORD LIBRARIANS, Palace Hotel, San Francisco, Oct. 5-9. Miss Doris Gleason, 510 N. Dearborn St., Chicago 10, Executive Secretary.

AMERICAN ASSOCIATION FOR THE SURGERY OF TRAUMA, Drake Hotel, Chicago, Oct. 1-3. Dr. James K. Stack, 700 North Michigan Blvd., Chicago, Secretary.

AMERICAN CANCER SOCIETY, New York, Nov. 2-6. Dr. Charles S. Cameron, 47 Beaver St., New York 4, Medical Director.

AMERICAN CLINICAL AND CLIMATOLOGICAL ASSOCIATION, The Homestead, Hot Springs, Va., Nov. 2-4. Dr. Marshall N. Fulton, 124 Waterman St., Providence 6, R. I., Secretary.

AMERICAN COLLEGE OF CARDIOLOGY, Hotel Statler, Cleveland, Ohio, Nov. 6-7. Dr. Philip Reichert, 480 Park Ave., New York 22, Secretary.

AMERICAN COLLEGE OF SURGEONS, Conrad Hilton Hotel, Chicago, Oct. 5-9 Dr. Michael L. Mason, 40 East Erie St., Chicago, Secretary.

AMERICAN DENTAL ASSOCIATION, Cleveland, Sept. 28-Oct. 1. Dr. Harold Hillenbrand, 222 E. Superior St., Chicago 11, General Secretary.

AMERICAN FRACTURE ASSOCIATION, Mission Inn, Riverside, Calif., Oct. 12-15 Dr. H. W. Wellmerling, 120 S. LaSalle St., Chicago 3, Secretary.

AMERICAN MEDICAL WRITERS' ASSOCIATION, Elk's Club, Springfield, Ill., Sept. 23. Dr. Harold Swanberg, 510 Maine St., Quincy, Ill., Secretary.

AMERICAN PUBLIC HEALTH ASSOCIATION, Hotel New Yorker and Hotel Statler, New York, Nov. 9-13. Dr. Reginald M. Atwater, 1790 Broadway, New York 19, Executive Secretary.

AMERICAN ROENTGEN RAY SOCIETY, Netherland Plaza, Cincinnati, Sept. 29-Oct. 2. Dr. Barton R. Young, Germantown Hospital, Philadelphia 44, Secretary.

AMERICAN SCHOOL HEALTH ASSOCIATION, Hotel New Yorker, New York, Nov. 9-13. Dr. A. O. DeWeese, 515 East Main St., Kent, Ohio, Secretary.

AMERICAN SOCIETY OF ANESTHESIOLOGISTS, Olympic Hotel, Seattle, Oct. 6-9. Dr. J. Earl Remlinger Jr., Room 1101, 188 W. Randolph St., Chicago, Secretary.

AMERICAN SOCIETY OF CLINICAL PATHOLOGISTS, Drake Hotel, Chicago, Oct. 12-16. Dr. Clyde G. Culbertson, 1040 W. Michigan St., Indianapolis, Secretary.

AMERICAN SOCIETY FOR THE STUDY OF ARTERIOSCLEROSIS, Hotel Knickerbocker, Chicago, Nov. 1-2. Dr. O. J. Pollak, P. O. Box 228, Dover, Del., Secretary.

AMERICAN SOCIETY OF TROPICAL MEDICINE AND HYGIENE, Brown Hotel, Louisville, Ky., Nov. 12-14. Dr. John E. Larsh Jr., University of North Carolina, School of Public Health, Chapel Hill, N. C., Secretary.

ASSOCIATION OF AMERICAN MEDICAL COLLEGES, Hotel Claridge, Atlantic City, N. J., Oct. 26-28. Dr. Dean F. Smiley, 185 N. Wabash Ave., Chicago 1, Secretary.

ASSOCIATION OF LIFE INSURANCE MEDICAL DIRECTORS OF AMERICA, Hotel Statler, New York, Oct. 14-16. Dr. Henry B. Kirkland, P. O. Box 594, Newark 1, N. J., Secretary.

ASSOCIATION OF MEDICAL ILLUSTRATORS, Sheraton Hotel, Baltimore, Oct. 5-7. Miss Evelyn J. Erickson, 1512 St. Antoine St., Detroit 26, Secretary.

ASSOCIATION OF MILITARY SURGEONS OF THE UNITED STATES, Hotel Statler, Washington, D. C., Nov. 8-11. Dr. R. R. Sayers, Armed Forces Institute of Pathology, Washington 25, D. C., Secretary.

ASSOCIATION OF STATE AND TERRITORIAL HEALTH OFFICERS, Washington Hotel, Washington, D. C., Nov. 4-7. Dr. John D. Porterfield, 306 Ohio Depts. Bldg., Columbus 15, Ohio, Secretary.

CENTRAL ASSOCIATION OF OBSTETRICIANS AND GYNECOLOGISTS, The Shamrock, Houston, Texas, Nov. 5-7. Dr. Harold L. Gainey, Suite 602, 116 South Michigan Ave., Chicago 3, Secretary.

CENTRAL NEUROPSYCHIATRIC ASSOCIATION, Claypool Hotel, Indianapolis, Oct. 15-17. Dr. Hamilton Ford, 112 North Blvd., Galveston, Texas, Secretary.

COLLEGE OF AMERICAN PATHOLOGISTS, Drake Hotel, Chicago, Oct. 12-16. Dr. Harry P. Smith, 203 N. Wabash Ave., Chicago 1, Secretary.

COLORADO STATE MEDICAL SOCIETY, Shirley-Savoy Hotel, Denver, Sept. 29-Oct. 2. Mr. Harvey T. Stethman, 835 Republic Bldg., Denver 2, Executive Secretary.

CONSTANTINIAN SOCIETY, Battery Park Hotel, Asheville, N. C., Oct. 18-21. Dr. C. F. Shook, Medical Director, Owens-Illinois Glass Company, Toledo 1, Ohio, Secretary.

DELAWARE, MEDICAL SOCIETY OF, Hotel du Pont, Wilmington, Oct. 12-14. Dr. Andrew M. Gehret, 822 North American Bldg., Secretary.

DISTRICT OF COLUMBIA, MEDICAL SOCIETY OF THE, Hotel Statler, Washington, Oct. 5-7. Mr. Theodore Wiprud, 1718 M St. N.W., Washington 6, Secretary.

GULF COAST CLINICAL SOCIETY, Admiral Semmes Hotel, Mobile, Ala., Oct. 15-16. Dr. William J. Atkinson Jr., 1217 Government St., Mobile 20, Ala., Secretary.

GULF COAST CONFERENCE ON INDUSTRIAL HEALTH, Shamrock Hotel, Houston, Tex., Oct. 1-3. Dr. Sidney Schnur, 411 Medical Arts Bldg., Houston 2, Tex., Co-Chairman.

INDIANA STATE MEDICAL ASSOCIATION, French Lick, Oct. 19-21. Mr. James A. Waggener, 23 E. Ohio St., Indianapolis 4, Executive Secretary.

INTERSTATE POST GRADUATE MEDICAL ASSOCIATION OF NORTH AMERICA, Palmer House, Chicago, Nov. 2-5. Dr. E. R. Schmidt, 1300 University Ave., Madison 5, Wisconsin, Secretary.

KANSAS CITY SOUTHWEST CLINICAL SOCIETY, Kansas City, Mo., Sept. 28-Oct. 1. Dr. W. H. Algie, 825 North 7th Street, Kansas City, Kans., Secretary.

KENTUCKY STATE MEDICAL ASSOCIATION, Brown Hotel, Louisville, Sept. 22-24. Dr. Bruce Underwood, 620 South Third St., Louisville 2, Secretary.

MICHIGAN STATE MEDICAL SOCIETY, Pantlind Hotel and Civic Auditorium, Grand Rapids, Sept. 23-25. Dr. L. Fernald Foster, 606 Townsend St., Lansing 15, Secretary.

MIDWESTERN SECTION OF AMERICAN FEDERATION FOR CLINICAL RESEARCH, Congress Hotel, Chicago, Oct. 29. Dr. Robert J. Glaser, 600 S. Kingshighway Blvd., St. Louis 10, Secretary.

MISSISSIPPI VALLEY MEDICAL SOCIETY, Springfield, Ill., Sept. 23-25. Dr. Harold Swanberg, 510 Maine St., Quincy, Ill., Secretary.

NATIONAL GASTROENTEROLOGICAL ASSOCIATION, Hotel Biltmore, Los Angeles, Oct. 12-17. Dr. A. Xerxes Rossien, 1819 Broadway, New York 23, Secretary.

NATIONAL PROCTOLOGIC ASSOCIATION, Congress Hotel, Chicago, Oct. 15-17. Dr. George E. Mueller, 59 East Madison St., Chicago 2, Secretary.

NATIONAL SOCIETY FOR CRIPPLED CHILDREN AND ADULTS, Palmer House, Chicago, Nov. 12-14. Mr. Lawrence J. Linck, 11 South LaSalle St., Chicago 3, Secretary.

NEUROSURGICAL SOCIETY OF AMERICA, The Broadmoor, Colorado Springs, Colo., Sept. 23-27. Dr. C. D. Hawkes, 22 North Manassas St., Memphis 5, Tenn., Secretary.

NEVADA STATE MEDICAL ASSOCIATION, Carter Hotel, Elko, Sept. 24-26. Dr. William A. O'Brien III, 505 Chestnut St., Reno, Secretary.

NEW ENGLAND OBSTETRICAL AND GYNECOLOGICAL SOCIETY, Hotel Somerset, Boston, Oct. 28. Dr. Carmi R. Alden, 270 Commonwealth Ave., Boston, Secretary.

NEW HAMPSHIRE MEDICAL SOCIETY, Equinox House, Manchester, Vt., Oct. 4-6. Dr. Deering G. Smith, 44 Chester St., Nashua, Secretary.

OKLAHOMA CITY CLINICAL SOCIETY, Oklahoma City, Oct. 26-29. Miss Alma F. O'Donnell, 512 Medical Arts Bldg., Oklahoma City 2, Executive Secretary.

OMAHA MID-WEST CLINICAL SOCIETY, Hotel Paxton, Omaha, Oct. 26-30. Miss Irene Rau, 1031 Medical Arts Bldg., Omaha 2, Executive Secretary.

OREGON STATE MEDICAL SOCIETY, Heathman Hotel, Portland, Oct. 14-17. Dr. Charles E. Littlehales, 833 S.W. Eleventh Ave., Portland 5, Secretary.

PENNSYLVANIA, MEDICAL SOCIETY OF THE STATE OF, William Penn Hotel, Pittsburgh, Sept. 21-24. Dr. Harold B. Gardner, 230 State St., Harrisburg, Secretary.

REGIONAL MEETINGS, AMERICAN COLLEGE OF PHYSICIANS:

WESTERN NEW YORK, Syracuse, Oct. 9. Dr. E. C. Reifenstein, 109 South Warren St., Syracuse 2, Governor.

OHIO, Dayton, Oct. 9. Dr. Charles A. Doan, Ohio State University College of Medicine, Columbus 10, Governor.

MONTANA-WYOMING, Missoula, Mont., Oct. 9-10. Dr. Harold W. Gregg, 127 West Park Street, Butte, Mont., Governor.

NEW ENGLAND—CONN., MAINE, MASS., N. H., R. I., VT.—Hartford, Conn., Oct. 28. Dr. John C. Leonard, 80 Seymour St., Hartford 15, Conn., Chairman.

NEW JERSEY, Trenton, Nov. 4. Dr. Edward C. Klein Jr., 73 Lincoln Park, Newark 2, Governor.

SOUTHEASTERN—Cuba, Alabama, Florida, Georgia, South Carolina—St. Simons, Sea Island, Ga., Oct. 16-17. Dr. Carter Smith, 384 Peachtree St. N.E., Atlanta 3, Ga., Chairman.

REGIONAL MEETINGS, AMERICAN RHEUMATISM ASSOCIATION:

OHIO AND WESTERN PENNSYLVANIA, Mayflower Hotel, Akron, Ohio, Nov. 11. Dr. Roger Q. Davis, 1405 Goodyear Blvd., Akron 5, Ohio, Chairman.

SOUTH CENTRAL BRANCH, AMERICAN UROLOGICAL ASSOCIATION, Kansas City, Mo., Sept. 20-23. Dr. Rex E. Van Duzen, 721 Medical Arts Bldg., Dallas, Texas, Chairman.

SOUTHERN MEDICAL ASSOCIATION, Municipal Auditorium, Atlanta, Ga., Oct. 26-29. Mr. C. P. Loranz, 1020 Empire Bldg., Birmingham 3, Ala., Secretary.

SOUTHWESTERN MEDICAL ASSOCIATION, El Conquistador Hotel, Tucson, Ariz., Oct. 29-31. Dr. C. C. Stapp, 800 Montana St., El Paso, Texas, Secretary.

SOUTHWESTERN SURGICAL CONGRESS, Hotel Utah, Salt Lake City, Sept. 21-23. Dr. C. R. Rountree, 1227 Classen St., Oklahoma City 3, Secretary.

TENNESSEE VALLEY MEDICAL ASSEMBLY, Read House Hotel, Chattanooga, Sept. 28-29. Dr. William G. Stephenson, 612 Medical Arts Bldg., Chattanooga 3, President.

VERMONT STATE MEDICAL SOCIETY, Equinox House, Manchester, Oct. 4-6. Dr. James P. Hammond, 542 Main St., Bennington, Secretary.

VIRGINIA, MEDICAL SOCIETY OF, Hotel Roanoke, Roanoke, Oct. 18-21. Mr. Robert I. Howard, 1105 W. Franklin St., Richmond 20, Executive Secretary.

WESTERN ORTHOPEDIC ASSOCIATION, Sun Valley, Idaho, Oct. 5-8. Dr. Forrest L. Flashman, 1301 Spring St., Seattle 4, Secretary.

WISCONSIN, STATE MEDICAL SOCIETY OF, Hotel Schroeder, Milwaukee, Oct. 5-8. Mr. Charles H. Crownhart, 704 East Gorham St., Madison 3, Secretary.

FOREIGN

ASSOCIATION OF SURGEONS OF GREAT BRITAIN AND IRELAND, Leeds, England, May 13-15, 1954. Dr. Henry W. S. Wright, 45 Lincoln's Inn Fields, London W.C.2, England, Honorary Secretary.

CANADIAN MEDICAL ASSOCIATION, Vancouver, B. C., Canada, June 18-22, 1954. Dr. T. C. Routley, 135 St. Clair Ave. W., Toronto 5, Ont., Canada, General Secretary.

CONGRESS OF INTERNATIONAL ANESTHESIA RESEARCH SOCIETY, Chateau Frontenac, Quebec, Canada, October 26-29. Dr. A. William Friend, 515 Nome Ave., Akron 20, Ohio, Chairman, Program Committee.

INTERIM MEETING, AERO MEDICAL ASSOCIATION, University of Brussels Medical School, Brussels, Belgium, Sept. 24-27. Dr. Andre Allard, Medical Director of SABENA, 145 Rue Royale, Brussels, Belgium, Secretary.

INTERNATIONAL CANCER CONGRESS, Sao Paulo, Brazil, July 23-29, 1954. Prof. A. Prudente, 171 rua Benjamin Constante, Sao Paulo, Brazil, President.

INTERNATIONAL CONFERENCE ON THROMBOSIS AND EMBOLISM, Basle, Switzerland, July 15-19, 1954. Dr. W. Merz, Chief Medical Officer, Gynecological Clinic, University of Basle, Basle, Switzerland, Hon. Secretary.

INTERNATIONAL CONGRESS ON GROUP PSYCHOTHERAPY, Toronto, Ont., Canada, Aug. 12-14, 1954. Dr. J. L. Moreno, Room 327, 101 Park Ave., New York 17, N. Y., U. S. A., Director of Organizing Committee.

INTERNATIONAL CONGRESS OF HYDROCLIMATISM AND THALASSOTHERAPY, Dubrovnik, Yugoslavia, May 8-16, 1954. Prof. C. Plavsic, Zeleni Venac 1, Belgrade, Yugoslavia, Secretary General.

INTERNATIONAL CONGRESS OF INTERNATIONAL COLLEGE OF SURGEONS, Sao Paulo, Brazil, April 26-May 2, 1954. Dr. Max Thorek, 1516 Lake Shore Drive, Chicago, Illinois, U.S.A., Secretary-General.

INTERNATIONAL CONGRESS ON MENTAL HEALTH, University of Toronto, Toronto, Ontario, Canada, Aug. 14-21, 1954. For information write: Executive Officer, International Congress on Mental Health, 111 St. George St., Toronto, Ontario, Canada.

INTERNATIONAL CONGRESS ON OBSTETRICS AND GYNECOLOGY, Geneva, Switzerland, July 26-31, 1954. Dr. H. de Watteville, Maternité Hôpital Cantonal, Geneva, Switzerland, President.

INTERNATIONAL CONGRESS OF PAEDIATRICS, Havana, Cuba, Oct. 12-17. Prof Felix Hurtado, 5a Avenue 124, Miramar, Havana, Cuba, President.

INTERNATIONAL CONGRESS OF PSYCHOLOGY, Montreal, Canada, June 7-12, 1954. For information write: Prof. H. S. Langfeld, International Union of Scientific Psychology, Eno Hall, Princeton University, Princeton, N. J., U. S. A.

INTERNATIONAL CONGRESS FOR PSYCHOTHERAPY, Zurich, Switzerland, July 21-24, 1954. Dr. H. K. Fierz, Theaterstrasse 12, Zurich 1, Switzerland, Secretary General.

INTERNATIONAL GERONTOLOGICAL CONGRESS, London and Oxford, England, July 12-22, 1954. Prof. R. E. Tunbridge, General Infirmary, Department of Medicine, The University, Leeds, England, President.

INTERNATIONAL LEPROSY CONGRESS, Madrid, Spain, Oct. 3-10. Dr. Felix Contreras, Moreto 15, Madrid, Spain, Secretary.

INTERNATIONAL OFFICE OF DOCUMENTATION OF MILITARY MEDICINE, Rome, Italy, Oct. 14-18. Colonel Medecin Prof. A. Campana, Office of the Ministero della Difesa-Esercito, Rome, Italy, Secretary.

JOURNEES MEDICALES, Paris, France, April 21-25, 1954. For information write: Secretariat of the Journees, 12, rue Pierre-Geofroix, Colombes (Seine) France.

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LATIN AMERICAN CONGRESS OF OBSTETRICS AND GYNECOLOGY, Buenos Aires, Argentina, Oct. 26-31.

LATIN AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY, Caracas, Venezuela, Feb. 21-25, 1954. Dr. Victorino Marquez Reveron, Centro Medico, Caracas, Venezuela, Secretary-General.

PACIFIC SCIENCE CONGRESS, Quezon City and Manila, Philippines, Nov. 16-28. Dr. Patrocinio Valenzuela, College of Pharmacy, University of the Philippines, Quezon City, Philippines, Secretary-General.

PAN AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY, Mexico, D.F., Mexico, Feb. 28-March 4, 1954.

PAN AMERICAN MEDICAL ASSOCIATION, International Medical Cruise Congress, S. S. Nieuw Amsterdam, Jan. 6-22, 1954. Dr. Joseph J. Eller, 745 Fifth Avenue, New York 22, N. Y., U. S. A., Executive Director.

PAN AMERICAN MEDICAL WOMEN'S ALLIANCE, Beekman Towers Hotel, New York, N. Y., Sept. 24-Oct. 1. Dr. Ina Marsh, 140 Linwood Ave., Buffalo, N. Y., U. S. A., Registration Chairman.

PERSIAN GULF MEDICAL SOCIETY, Dhahran, Saudi Arabia, Dec. 7-8. Dr. N. J. Conan Jr., Department of Internal Medicine, Arabian American Oil Company, Dhahran, Saudi Arabia, Secretary.

REGIONAL MEETING, AMERICAN COLLEGE OF PHYSICIANS, San Juan, Puerto Rico, Oct. 15-17. Dr. R. Rodriquez-Molina, Veterans Administration Center, San Juan, Puerto Rico, Governor.

SECTIONAL MEETING, AMERICAN COLLEGE OF SURGEONS, London, England, May 17-19, 1954. Dr. Michael L. Mason, 40 East Erie St., Chicago 11, Ill., U. S. A., Secretary.

EXAMINATIONS AND LICENSURE

BOARDS OF MEDICAL EXAMINERS

ALABAMA: *Examination.* Montgomery, June 22-24, 1954. Sec., Dr. D. G. Gill, 537 Dexter Ave., Montgomery.

ARIZONA:* *Examination.* Phoenix, Oct. 22-23. *Endorsement.* Phoenix, Oct. 24. Executive Secretary, Mr. Robert Carpenter, 401 Security Bldg., Phoenix.

ARKANSAS:* *Examination.* Little Rock, Nov. 5-6. Sec., Dr. Joe Verser, Harrisburg.

COLORADO:* *Reciprocity.* Denver, Oct. 13. Final date for filing application was Sept. 14. *Examination.* Denver, Dec. 15-16. Final date for filing application is Nov. 14. Executive Secretary, Miss Beulah H. Hudgens, 831 Republic Bldg., Denver 2.

CONNECTICUT:* *Regular. Examination.* New Haven, Nov. 10-11. Reciprocity actions are taken continuously. Secretary to the Board, Dr. Creighton Barker, 160 St. Ronan St., New Haven. *Homeopathic.* Derby, Nov. 10-11. Sec., Dr. Donald A. Davis, 38 Elizabeth St., Derby.

DELAWARE: Dover, Jan. 12-14, 1954. Sec., Dr. Joseph S. McDaniel, 229 South State St., Dover.

FLORIDA:* *Examination.* Jacksonville, Nov. 22-24. Sec., Dr. Homer L. Pearson, 901 N.W. 17th St., Miami 36.

GUAM: The Commission on Licensure will meet whenever a candidate appears or submits his credentials. Director of Medical Services, Guam Memorial Hospital, Agana.

IDAHO: Boise, Jan. 11-13, 1954. Executive Secretary, Mr. Armand L. Bird, 364 Sonna Bldg., Boise.

ILLINOIS: Chicago, Oct. 6-8. Superintendent of Registration, Mr. Fredric B. Selcke, State House, Springfield.

IOWA:* *Examination.* Des Moines, Dec. 7-9. Sec., Dr. M. A. Royal, 506 Fleming Bldg., Des Moines.

KANSAS: Topeka, Dec. 9-10. Sec., Dr. O. W. Davidson, 864 New Brotherhood Bldg., Kansas City.

MAINE: Portland, Nov. 12-13. Sec., Dr. Adam P. Leighton, 192 State Street, Portland.

MARYLAND: *Examination.* Baltimore, Dec. 8-11. Sec., Dr. Lewis P. Gundry, 1215 Cathedral St., Baltimore 1.

MICHIGAN:* *Examination.* Lansing, Oct. 14-16. Sec., Dr. J. Earl McIntyre, 202-4 Hollister Bldg., Lansing 8.

MINNESOTA:* Minneapolis, Oct. 20-22. Sec., Dr. E. M. Jones, 230 Lowry Medical Arts Bldg., St. Paul 2.

MISSISSIPPI: *Reciprocity.* Jackson, December. Assistant Secretary, Dr. R. Whitfield, Old Capitol, Jackson 113.

MISSOURI: Kansas City, Oct. 22-24. Ex. Sec., Mr. John A. Hailey, Box 4, State Capitol Bldg., Jefferson City.

MONTANA: Helena, Oct 5. Sec., Dr. S. A. Cooney, 214 Power Block, Helena.

NEBRASKA:* *Examination.* Omaha, June 1954. Director, Mr. Husted K. Watson, 1009 State Capitol Bldg., Lincoln.

NEVADA:* Reno, Oct. 6. Sec., Dr. George H. Ross, 112 Curry St., Carson City.

NEW JERSEY: *Examination.* Trenton, Oct. 20-23. Sec., Dr. E. S. Hallinger, 28 W. State St., Trenton.

NEW MEXICO:* Oct. 12-13, Santa Fe. Sec., R. C. Derbyshire, 227 E. Palace Ave., Sante Fe.

NEW YORK: *Examination.* New York, Syracuse, Buffalo and Albany, Oct. 6-9. Sec., Dr. Stiles D. Ezell, 23 South Pearl St., Albany.

NORTH CAROLINA: *Reciprocity.* Asheville, Oct. 12. Sec., Dr. Joseph J. Combs, 716 Professional Bldg., Raleigh.

NORTH DAKOTA: *Examination.* Grand Forks, Jan. 6-9, 1954. *Reciprocity.* Grand Forks, Jan. 9. Sec., Dr. C. J. Glaspel, Grafton.

OHIO: *Examination.* Columbus, December. *Reciprocity.* Columbus, Oct. 6. Sec., Dr. H. M. Platter, 21 W. Broad St., Columbus 15.

OREGON:* *Reciprocity.* Portland, Oct. 16-17. Sec., Dr. Wilmot C. Foster, 609 Failing Bldg., Portland 4.

PENNSYLVANIA: *Examination.* Philadelphia, January 1954. Acting Secretary, Mrs. Margaret G. Steiner, Box 911, Harrisburg.

RHODE ISLAND:* *Examination.* Providence, Oct. 1-2. Administrator of Professional Regulation, Mr. Thomas B. Casey, 366 State Office Bldg., Providence.

SOUTH CAROLINA: *Reciprocity.* Columbia, Sept. 21. Sec., Mr. N. B. Heyward, 1329 Blanding St., Columbia.

SOUTH DAKOTA:* *Examination.* Sioux Falls, Jan. 19-20, 1954. Reciprocity can be obtained at any time between meetings of board. Executive Secretary, Mr. John C. Foster, 300 First National Bank Bldg., Sioux Falls.

TENNESSEE:* *Examination.* Memphis, Sept. 30-Oct. 1. Sec., Dr. H. W. Qualls, 1635 Exchange Bldg., Memphis.

TEXAS: Fort Worth, Nov. 12-14. Reciprocity applicants must be completed 30 days prior to the meeting date. Sec., Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth 2.

WEST VIRGINIA: Charleston, Oct. 19. Sec., Dr. N. H. Dyer, State Office Bldg., Charleston 5.

WISCONSIN:* *Reciprocity.* Madison, October. *Examination and Reciprocity.* Madison, Jan. 12-14, 1954. Sec., Dr. A. G. Koehler, 46 Washington Blvd., Oshkosh.

WYOMING: Cheyenne, Oct. 5. Sec., Dr. Franklin D. Yoder, State Office Bldg., Cheyenne.

BOARDS OF EXAMINERS IN THE BASIC SCIENCES

ALASKA: On application. Juneau or other towns in Territory as decided by Board. *Reciprocity.* On application. Sec., Dr. C. Earl Albrecht, Box 1931, Juneau.

ARKANSAS: *Examination.* Little Rock, Oct. 6-7. Sec., Mr. Louis E. Gebauer, 1002 Donaghey Bldg., Little Rock.

CONNECTICUT: *Examination.* New Haven, Oct. 10. Address: State Board of Healing Arts, 52 Whitney Ave., New Haven 10.

DISTRICT OF COLUMBIA: *Examination.* Washington, Oct. 19-20. Sec., Dr. Daniel L. Seckinger, 4130 E. Municipal Bldg., Washington.

FLORIDA: *Examination.* Gainesville, Nov. 7. Sec., Mr. M. W. Emmel, Box 340, University of Florida, Gainesville.

IOWA: *Examination.* Des Moines, Oct. 13. Sec., Dr. Ben H. Peterson, Coe College, Cedar Rapids.

MICHIGAN: *Examination.* Detroit and Ann Arbor, Oct. 9-10. Sec., Mrs. Ann Baker, 410 W. Allegan St., Lansing.

MINNESOTA: *Examination.* Minneapolis, Oct. 6-7. Sec., Dr. Raymond N. Bieter, 105 Millard Hall, University of Minnesota, Minneapolis 14.

NEBRASKA: *Examination.* Omaha, Oct. 6-7. Director, Mr. Husted K. Watson, 1009 State Capitol Bldg., Lincoln.

NEVADA: *Examination.* Reno, Oct. 6. Sec., Dr. Donald G. Cooney, Box 9005, University Station, Reno.

NEW MEXICO: *Examination.* Santa Fe, Oct. 18. Sec., Mrs. Marguerite Cantrell, Box 1592, Santa Fe.

SOUTH DAKOTA: *Examination.* Vermillion, Dec. 4-5. Sec., Dr. Gregg M. Evans, 310 E. 15th St., Yankton.

TENNESSEE: *Examination.* Memphis, Sept. 23-24. Sec., Dr. O. W. Hyman, 874 Union Ave., Memphis 3.

TEXAS: *Examination.* Dallas and Galveston, October. Sec., Bro. Raphael Wilson, 407 Perry-Brooks Bldg., Austin.

WISCONSIN: *Examination.* Madison, Sept. 19. Final date for filing application is Sept. 11. Milwaukee, Dec. 5. Final date for filing application is Nov. 27. Sec., Dr. W. H. Barber, 621 Ransom St., Ripon.

* Basic Science Certificate required.

DEATHS

Woglom, William Henry ✦ Teaneck, N. J.; born in Brooklyn Sept. 13, 1879; Columbia University College of Physicians and Surgeons, New York, 1901; assistant in pathology at the Long Island College Hospital in Brooklyn, 1907-1908; pathologist and bacteriologist for the department of health of New York City, 1908-1909; from 1909 to 1912 assistant in cancer research at Columbia and in 1911-1912 served as assistant to the director of the Imperial Cancer Research Fund in London; from 1912 to 1917 assistant professor of cancer research, Institute of Cancer Research at Columbia University College of Physicians and Surgeons, where he was an associate professor from 1917 to 1946, when he became professor emeritus; in 1936 received a medal from Columbia University for conspicuous alumni service; an Affiliate Fellow of the American Medical Association; in 1937 president of the American Association for Cancer Research, of which he was at one time vice-president, for many years a member of the council, and secretary-treasurer; past president of the New York Pathological Society; member of the American Association for the Advancement of Science, Society of Experimental Biology and Medicine, and the Holland Society; an associate of the Royal Photographic Society of Great Britain; member of the scientific advisory committee of the International Cancer Research Foundation from 1941 to 1946; author of "Discoverers for Medicine" and several monographs on cancer; translated several technical books to English from French and German, including "Riddle of Cancer"; for many years editor of the *Journal of Cancer Research*; died in Columbia-Presbyterian Medical Center in New York City Aug. 8, aged 73, of cancer.

Wheaton, Clarence Loyd ✦ Chicago; born in Fort Pembina, N. D., May 27, 1873; Bellevue Hospital Medical College, New York, 1896; an Associate Fellow of the American Medical Association and member of its House of Delegates in 1909; member of the American College of Chest Physicians and the American Trudeau Society; past president of the North Side Branch of the Chicago Medical Society and the Chicago Tuberculosis Society; member of the board of directors of the National Tuberculosis Association; at one time vice-president of the Tuberculosis Institute of Chicago and Cook County of which he was honorary director; formerly instructor in medicine at Rush Medical College; served during World War I as president of the tuberculosis board of the U. S. Army at Camp Grant, Ill.; on the staffs of the City of Chicago Municipal Tuberculosis Sanitarium, Evangelical Deaconess Hospital, and the Illinois Masonic Hospital, where he died June 4, aged 80, of arteriosclerotic heart disease.

Johnson, Leighton Foster ✦ Boston; born in Hingham, Mass., Nov. 30, 1890; Boston University School of Medicine, 1915; professor of otolaryngology at his alma mater; specialist certified by the American Board of Otolaryngology; member of the American Academy of Ophthalmology and Otolaryngology, American Laryngological, Rhinological and Otological Society, New England Otolaryngological Society, and the American Broncho-Esophagological Association; served during World War I; fellow of the American College of Surgeons; chief of otolaryngological service, Massachusetts Memorial Hospitals; consultant in otolaryngology, Norwood (Mass.) Hospital, Westboro (Mass.) State Hospital, and Massachusetts Hospital School in Canton; consultant in laryngology, Cape Cod Hospital, Hyannis, and Martha's Vineyard Hospital in Oak Bluffs; consultant, Burbank Hospital in Fitchburg, Mass.; died in Wellfleet July 21, aged 62.

Pugliese, Francis Michael ✦ Wilkes-Barre, Pa.; born in Calimera, Italy, Feb. 7, 1899; University of Pennsylvania School of Medicine, Philadelphia, 1923; specialist certified by the American Board of Surgery; fellow of the American College of Surgeons; member of the Alumni Association of Mayo Foundation; at one time county treasurer; lieutenant colonel in the U. S. Army during World War II, serving in the European Theatre as chief of surgery of a Station Hospital; formerly a fellow in surgery of the Mayo Foundation, Rochester, Minn.; consultant at St. Joseph's Hospital in Hazleton; affiliated with Nanticoke (Pa.) State Hospital, Pittston (Pa.) Hospital, and the Mercy Hospital, where he died Aug. 13, aged 54, of cerebral hemorrhage and hypertensive cardiorenal disease.

Braden, Albert Henry ✦ Houston, Texas; born in Bernardo, Texas, Oct. 30, 1886; University of Texas School of Medicine, Galveston, 1914; specialist certified by the American Board of Pathology; member of the American College of Allergists, American Society of Clinical Pathologists, International Correspondence Society of Allergists, Post Graduate Medical Assembly of South Texas, Southern Medical Association, Southwest Allergy Forum, and the American Association for the Advancement of Science; past president of the Texas Society of Pathologists; for many years affiliated with St. Joseph's Hospital; died in St. Joseph's Hospital July 22, aged 66.

Hohf, Silas Matthew ✦ Yankton, S. D.; Illinois Medical College, Chicago, 1897; Northwestern University Medical School, Chicago, 1903; fellow of the American College of Surgeons; for 29 years an instructor on the faculty of the University of South Dakota School of Medicine; member of the board of education from 1912 to 1928; in 1928 president of the South Dakota State Medical Association; served during World War I; affiliated with Sacred Heart Hospital, where he died June 28, aged 80, of bronchopneumonia and cerebral arteriosclerosis.

Allen, Edward Carson ✦ Newton, Mass.; Tufts College Medical School, Boston, 1939; certified by the National Board of Medical Examiners; specialist certified by the American Board of Anesthesiology; member of the American Society of Anesthesiologists; served during World War II; affiliated with Faulkner Hospital in Boston; died July 14, aged 38, of a heart attack.

Allen, Joseph Randolph ✦ Los Angeles; Northwestern University Medical School, Chicago, 1906; died May 14, aged 78, of cerebral thrombosis and general arteriosclerosis.

Arnold, Laurie James, Lake City, Fla.; North Carolina Medical College, Davidson, 1905; died May 5, aged 75, of coronary occlusion.

Barnaby, Charles W., Daytona Beach, Fla.; Detroit College of Medicine, 1894; died in Halifax District Hospital May 28, aged 87, of uremia and pyelonephritis.

Black, Emil Claudius ✦ San Diego, Calif.; University of Nebraska College of Medicine, Omaha, 1903; an Associate Fellow of the American Medical Association; affiliated with Mercy and San Diego County General hospitals; died July 2, aged 78, of chronic myocarditis and arteriosclerosis.

Boynton, Lloyd V. ✦ Peoria, Ill.; St. Louis University School of Medicine, 1906; an Associate Fellow of the American Medical Association; affiliated with St. Francis Hospital, where he was past president of the staff; died in Proctor Hospital July 12, aged 73, of acute coronary thrombosis.

Briscoe, Hugh Alan ✦ Akron, Ohio; University of Cincinnati College of Medicine, 1918; died in Broward General Hospital, Fort Lauderdale, Fla., June 19, aged 61, of cerebral hemorrhage and hypertension.

Brownell, Emily Margaret Baer ✦ San Diego, Calif.; University of Illinois College of Medicine, Chicago, 1936; past president of the San Diego Branch of the American Medical Women's Association; affiliated with La Mesa (Calif.) Hospital, Mercy, Quintard, and San Diego County General hospitals; died June 10, aged 43, of massive myocardial infarction.

✦ Indicates Member of the American Medical Association.

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Busching, Howard Ellsworth, Wyoming, Ohio; University of Cincinnati College of Medicine, 1920; died in Bethesda Hospital, Cincinnati, July 7, aged 58, of coronary occlusion.

Buschmann, Bronson, Seattle; University of Pennsylvania School of Medicine, Philadelphia, 1947; resident in surgery, King County Hospital; served during World War II; died June 28, aged 29.

Campbell, Colin Clyde, Ashley, N. D.; University of Toronto Faculty of Medicine, Toronto, Ontario, Canada, 1901; served as county health officer; died in Rome, N. Y., July 9, aged 77, of coronary occlusion.

Case, Edward Franklin ✦ Seattle; Creighton University School of Medicine, Omaha, 1934; served during World War II; died in Doctors Hospital recently, aged 49, of coronary occlusion with infarction of the myocardium.

Curtiss, Fred Decker, Kenosha, Wis.; Chicago Homeopathic Medical College, 1901; died in Kenosha Hospital April 22, aged 76, of ruptured aneurysm of the abdominal aorta.

Dame, Louis Paul ✦ Rockford, Ill.; University of Illinois College of Medicine, Chicago, 1917; formerly medical missionary in Arabia; served during World War I; affiliated with St. Anthony Hospital, where he died July 2, aged 66, of gastrointestinal and pulmonary hemorrhage and cirrhosis of the liver.

Danglade, James Harold ✦ Kansas City, Mo.; University of Kansas School of Medicine, Kansas City, 1926; assistant professor of medicine at his alma mater; specialist certified by the American Board of Internal Medicine; fellow of the American College of Physicians; served during World War II; affiliated with St. Luke's Hospital, where he was past vice-president of the executive staff; died in Veterans Administration Hospital Aug. 4, aged 52, of hemorrhage from esophageal varices.

Drake, Clifford Cochrane, Roanoke, Va.; University of Nashville (Tenn.) Medical Department, 1898; died May 18, aged 80, of carcinoma of the prostate.

Fuller, Tilman, Oklahoma City, Okla. (licensed in Oklahoma under Act of 1908); died July 16, aged 80, of arteriosclerotic heart disease.

Gee, Lemuel Edmond ✦ Broken Bow, Okla. (licensed in Oklahoma under the Act of 1908); served as city health officer; at one time health officer of Hunt County, Texas; for many years physician for the Dierks Lumber and Coal Company; died in DeQueen (Okla.) General Hospital July 14, aged 74, of coronary thrombosis.

Griffith, Martin E. ✦ Monessen, Pa.; Jefferson Medical College of Philadelphia, 1885; served two terms as coroner; company doctor for the Pittsburgh Steel Company for 25 years; affiliated with the Memorial Hospital in Monongahela, where he died May 1, aged 92.

Haig, Thomas Raymond ✦ Carlsbad, Calif.; Stanford University School of Medicine, San Francisco, 1922; formerly clinical instructor in orthopedics at his alma mater; for many years practiced in Sacramento, where he was a trustee of the Sutter General Hospital; died suddenly in San Francisco July 13, aged 57, of coronary thrombosis.

Harris, Dean Wilson ✦ Fremont, Mich.; Eclectic Medical College, Cincinnati, 1922; past president of the Newaygo County Medical Society; on the staff of the Gerber Memorial Hospital; died in St. Mary's Hospital, Grand Rapids, July 6, aged 60, of left bundle branch block, recurrent coronary occlusion with infarction, and cholelithiasis.

Hedrick, Donald William ✦ Tampa, Fla.; University of Michigan Medical School, Ann Arbor, 1928; specialist certified by the American Board of Orthopaedic Surgery; member of the Clinical Orthopaedic Society and the American Academy of Orthopaedic Surgeons; fellow of the American College of Surgeons; on the staffs of St. Joseph's Hospital and Tampa Municipal Hospital, where he died May 27, aged 50, of coronary arteriosclerosis.

Hoover, James J. D. ✦ Terre Haute, Ind.; Meharry Medical College, Nashville, Tenn., 1908; owner and superintendent of the Hoovers' Sanitarium; died April 21, aged 71, of coronary insufficiency.

Huson, William Joseph ✦ Covington, Ga.; University of Georgia School of Medicine, Augusta, 1924; served during World War I; died in Atlanta June 27, aged 58, of cardiorespiratory failure.

Jamison, Alfred, Marks, Miss.; Memphis (Tenn.) Hospital Medical College, 1908; died July 11, aged 72, of angina pectoris.

Jones, Henry Durst ✦ Schleswig, Iowa; State University of Iowa College of Medicine, Iowa City, 1903; died July 4, aged 73, of cerebral arteriosclerosis.

Joyce, Edgar Collins, Tuckahoe, N. Y.; Columbia University College of Physicians and Surgeons, New York, 1895; served during Spanish-American War and World War I; formerly on the staffs of St. Vincent's and Fordham hospitals in New York; died in Veterans Administration Hospital in Beacon, July 27, aged 81, of arteriosclerotic heart disease.

Kirksey, James Jackson ✦ Morganton, N. C.; University of Pennsylvania School of Medicine, Philadelphia, 1921; died in the Grace Hospital May 16, aged 60, of an acute heart attack.

Kirkwood, Robert Carnahan, Hampton, Va.; Jefferson Medical College of Philadelphia, 1908; died in Veterans Administration Hospital May 24, aged 66, of coronary thrombosis.

Kushell, Maria Dora, New York City; University of Kharkov Faculty of Medicine, Russia, 1918; affiliated with Gouverneur and Manhattan General hospitals; died in Beth Israel Hospital July 16, aged 59, of cancer.

Laurent, Francis Victor, Maitland, Fla.; Baltimore Medical College, 1908; died May 20, aged 76, of bronchopneumonia and carcinoma of the prostate.

Leitschuh, Linus Frederick ✦ Winsted, Minn.; University of Minnesota Medical School, Minneapolis, 1938; served during World War II; died in St. Mary's Hospital, Minneapolis, June 28, aged 41, of hepatitis and esophageal varices.

McLarand, Kenneth Carl ✦ Santa Monica, Calif.; State University of Iowa College of Medicine, Iowa City, 1936; on the staffs of Long Beach (Calif.) Hospital and Santa Monica Hospital; died April 16, aged 47.

McLeod, James Lawrence ✦ Grand Rapids, Minn.; University of Manitoba Faculty of Medicine, Winnipeg, Canada, 1926; state senator in 1935-1936; affiliated with Itasca Memorial Hospital, where he died June 8, aged 52, of cardiac decompensation and pulmonary embolism.

Malick, Ada Luella, Piqua, Ohio; College of Physicians and Surgeons of Chicago, School of Medicine of the University of Illinois, 1900; died June 30, aged 78, of heart disease.

Morgenthaler, Sophia, Daytona Beach, Fla.; New York Medical College and Hospital for Women, Homeopathic, New York, 1894; died June 25, aged 56, of organic heart disease.

Morris, Joseph Bowman, Punxsutawney, Pa.; Jefferson Medical College of Philadelphia, 1886; died in Adrian Hospital May 25, aged 92, as the result of a fall.

Murchie, John Talmadge, Portsmouth, Ohio; Rush Medical College, Chicago, 1921; died in Broward General Hospital in Fort Lauderdale, Fla., May 26, aged 60, of bronchogenic carcinoma of the right lung.

Neves, Carl Albert ✦ Custer, S. D.; Atlanta College of Physicians and Surgeons, 1910; formerly manager, Veterans Administration Center, Hot Springs, died in Veterans Administration Hospital in Fort Snelling, Minn., June 28, aged 66, of bronchogenic carcinoma of the right lung.

Neville, J. C. ✦ Register, Ga.; Medical College of Georgia, Augusta, 1900; on the staff of Bulloch County Hospital, Statesboro, where he died June 22, aged 80, of injuries received in an automobile accident.

Newsom, Goebel William, Fort Pierce, Fla.; Kansas City (Mo.) University of Physicians and Surgeons, 1929; died May 15, aged 53, of arteriosclerotic heart disease and hypertension.

O'Neill, Silverton Hollingsworth ✦ Blue Hill, Neb.; College of Physicians and Surgeons, Baltimore, 1907; died June 3, aged 76.

Pember, Clyde Harry, Van Nuys, Calif.; American Medical College, St. Louis, 1898; served during World War I and received the Distinguished Service Cross, Croix de Guerre and the Purple Heart; served on the staffs of various Veterans Administration hospitals; died in Veterans Administration Center in Los Angeles, June 22, aged 80, of coronary thrombosis.

Perry, Henry Ernest ✦ Newberry, Mich.; Michigan College of Medicine and Surgery, Detroit, 1897; Northwestern University Medical School, Chicago, 1904; past president of the Michigan State Medical Society, Luce County Medical Society, and the Upper Peninsula Medical Society; veteran of the Spanish-American War; formerly affiliated with the Newberry State Hospital; served in the state legislature, 1933-1934; died in Lakeland, Fla., July 9, aged 87, of uremia and arteriosclerotic heart disease.

Putnam, John Lancaster, Lebanon, Ky.; Kentucky School of Medicine, Louisville, 1906; served during World War I; affiliated with Mary Immaculate Hospital, where he died July 7, aged 70, of coronary thrombosis.

Quinn, William Blanck, Jackson Heights, N. Y.; Georgetown University School of Medicine, Washington, D. C., 1926; served during World War II; affiliated with Manhattan General Hospital in New York City; died July 15, aged 52, of a heart attack.

Ramsdell, Robert Leroy ✦ Austin, Texas; University of Texas School of Medicine, Galveston, 1913; past president of the Dallas County Medical Society; formerly on the staff of the Austin State Hospital; affiliated with Central State Hospital at Norman, Okla.; died in Ardmore, Okla., July 5, aged 71, of cerebral hemorrhage.

Randall, Auvigne Mason ✦ Ashby, Minn.; Minneapolis College of Physicians and Surgeons, medical department of Hamline University, 1903; on the staff of St. Luke's Hospital; died June 20, aged 72, of coronary occlusion.

Reeve, Edward A. Tanner ✦ Elbow Lake, Minn.; Minneapolis College of Physicians and Surgeons, medical department of Hamline University, 1897; died June 5, aged 79, of carcinoma of the sigmoid.

Rice, Thompson R. ✦ Petersburg, Ind.; Hahnemann Medical College and Hospital of Philadelphia, 1892; an Associate Fellow of the American Medical Association; died in St. Mary's Hospital, Evansville, May 9, aged 88, of intestinal obstruction and arteriosclerotic heart disease.

Richardson, John Purver ✦ Ellensburg, Wash.; Medical College of the State of South Carolina, Charleston, 1919; fellow of the American College of Surgeons; administrator of the Ellensburg General Hospital, where he died July 4, aged 60, of heart failure.

Rooney, Eugene Francis ✦ New York City; St. Louis University School of Medicine, 1923; specialist certified by the American Board of Pediatrics; served during World War I; director of pediatrics and past president of Misericordia Hospital; affiliated with St. Vincent's Hospital; past president of New York Foundling and St. Clare's hospitals; died in East Northport July 16, aged 57, of acute coronary occlusion.

Rosenfeld, Sydney Cyril ✦ Chicago; Chicago Medical School, 1937; affiliated with the American Hospital, where he died July 25, aged 46, of multiple sclerosis and terminal bronchopneumonia.

Shafer, John Walter ✦ Lafayette, Ind.; Rush Medical College, Chicago, 1900; served during World War I; for many years member of the city council; president of the Morocco State Bank; affiliated with St. Elizabeth's Hospital and the Lafayette Home Hospital, where he died July 10, aged 80, of coronary occlusion.

Smith, Francis Michael ✦ Vicksburg, Miss.; Memphis (Tenn.) Hospital Medical College, 1902; for many years county health officer; formerly health officer of Pine Bluff and of Jefferson County in Arkansas; died July 22, aged 74, of arteriosclerotic heart disease.

Smith, Morton Hartman, Hughesville, Pa.; Medico-Chirurgical College of Philadelphia, 1897; died in Muncy (Pa.) Valley Hospital July 5, aged 79, of coronary occlusion.

Sprague, Emory Russell, Rochester, N. Y.; Hahnemann Medical College and Hospital of Philadelphia, 1904; died July 4, aged 75, of coronary occlusion.

Tinley, Mary Louise ✦ Council Bluffs, Iowa; Omaha Medical College, 1895; member of the American Academy of General Practice; affiliated with Jennie Edmundson Hospital and the Mercy Hospital; named "Woman of the Year" in March, 1953, by the Women's Division of the Chamber of Commerce, and was awarded a gold inscribed bracelet; died July 24, aged 84, of heart disease.

Townsend, Junius M., Morton, Miss.; Memphis (Tenn.) Hospital Medical College, 1904; died July 13, aged 74, of injuries received in an automobile accident.

Turchi, John Dominic ✦ Philadelphia; University of Pennsylvania School of Medicine, Philadelphia, 1923; affiliated with Misericordia Hospital; died in the Graduate Hospital of the University of Pennsylvania July 13, aged 53, of pneumonia and cerebral vascular accident.

Ullman, Julius ✦ Buffalo; University of Buffalo School of Medicine, 1893; an Associate Fellow of the American Medical Association; formerly on the faculty of his alma mater; fellow of the American College of Physicians; died Aug. 14, aged 81.

Waterbury, D. Elliot, Melbourne, Fla.; Chicago College of Medicine and Surgery, 1913; died July 23, aged 91, of cerebral hemorrhage and arteriosclerosis.

Wedekind, Robert ✦ Louisville, Ky.; Hospital College of Medicine, Louisville, 1897; died recently, aged 82, of myocarditis and arteriosclerosis.

Weimer, Edgar Stanley ✦ Clearwater, Fla.; University of Pittsburgh School of Medicine, 1910; specialist certified by the American Board of Ophthalmology; fellow of the American College of Surgeons; served during World War I; formerly affiliated with the U. S. Public Health Service; died in St. Petersburg July 5, aged 68, of chronic nephritis.

Whitbeck, Sherwood Volkert ✦ Hudson, N. Y.; Columbia University College of Physicians and Surgeons, New York, 1901; fellow of the American College of Surgeons; on the staff of the Columbia Memorial Hospital, where he died July 23, aged 73, of Parkinson's disease.

Williams, Aubrey H. ✦ Denver; Gross Medical College, Denver, 1896; formerly on the faculty of the Denver and Gross College of Medicine, and the University of Colorado School of Medicine; died in the Mercy Hospital July 15, aged 83, of fracture of surgical neck of right femur.

Wilson, Robert Patterson Clark, Platte City, Mo.; Ensworth Medical College, St. Joseph, Mo., 1900; superintendent of the Missouri State School in Marshall; died in Marshall July 12, aged 77, of coronary occlusion.

Yates, Oscar Raymond ✦ Suffolk, Va.; Medical College of Virginia, Richmond, 1916; past president and vice-president of the Seaboard Medical Association; formerly member and chairman of the school board; active in Boy Scout work and in December, 1948, received the Silver Beaver Award presented by the Old Dominion Area Council of Boy Scouts, of which he was a member; served during World War I; chief of staff at Louise Obici Memorial Hospital, where he died July 15, aged 62, of coronary thrombosis.

York, Charles E. ✦ Chicago; the Hahnemann Medical College and Hospital, Chicago, 1917; affiliated with Illinois Masonic Hospital, where he died July 21, aged 64, of acute myocardial infarction.

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FOREIGN LETTERS

AUSTRALIA

The Commonwealth Medical Benefits Scheme.—The final phase of Australia's answer to the socialized medical schemes of Britain and New Zealand was put into operation July 1. The underlying principle of the Commonwealth Medical Benefits Scheme is the support from government funds of voluntary insurance against the costs of professional services. The Commonwealth Health Department has published a schedule of payments that will be made for a comprehensive range of medical services. A "medical benefits" organization must make a benefit available that at least equals the government subsidy.

The patient pays the professional fee, then sends the receipt to the medical benefits organization, which reimburses the patient for about 90% of the fee. The reimbursement is in two halves. One-half is the organization's own benefit and the other half is the government subsidy for the services involved. The organization then, in turn, files a claim with the government fund for the subsidy. No government subsidy is payable, however, unless the patient is a "financial" subscriber to a medical benefits fund. An aspect of this scheme that differs from American prepaid plans is that the Australian scheme covers the cost of "office" medical consultations and domiciliary visits by the general practitioner.

Advantages of this scheme are said to be as follows: 1. It encourages the principle of self-help. 2. It makes use of voluntary organizations to provide the administrative machinery. 3. It preserves the confidential doctor-patient relationship. 4. There is free choice by the patient of hospital, general practitioner, and specialist. The only duty imposed on the physician is to make out an itemized account in accordance with the government fee schedule, to facilitate payment of claims both by the organizations and the government. Unlike the United States where Blue Shield and Blue Cross plans are separate, most organizations in Australia cover both hospital and medical benefits. The largest organization in Australia is the "Medical Benefits Fund of Australia," which is sponsored by the medical profession. All medical benefits funds must be approved by the government, and a list of approved organizations is published in the daily press.

Following is an example of how the medical and hospital benefits scheme works: For purposes of monetary comparison, nine shillings in Australian money is equivalent in exchange value to one United States dollar. In relation to the costs of living and services, however, average costs and incomes are about double in America compared to Australia. Mr. A. has a wife and three children under 17 years of age. He takes out a maximum policy for both hospital and medical benefits. He pays 3s. (33 cents) each week for each of the hospital and medical benefits, totaling 66 cents weekly. This covers himself, his wife, and an unlimited number of children under 17, so that if his wife should have twins, his premium will not increase. If his wife consults a physician of her choice at his office, she will pay the physician 15 shillings ($1.66). She sends the receipted account (which reads "consultation 15s.") to the medical benefits fund. She receives a refund of 13s. 6d. from the fund, made up of 7s. 6d. from the fund, and 6s. from the government subsidy. If she is found to require an appendicectomy, she will go to a hospital of her choice where she will occupy a room at a cost of £14.14 per week. She stays in hospital two weeks. The account for her hospital room will be £29.8. Before she receives the account, however, it will have had 8s. per day deducted from it. This is a Commonwealth subsidy, which originated in 1946. It is paid directly to the hospital and the payment is made irrespective of whether the patient is a contributor to the medical benefits fund or not. This leaves a balance of £23.16 to be paid by Mrs. A. When she pays the account, she receives a certificate of hospitalization from the hospital, which she presents, with the receipted account, to the medical benefits fund. The fund repays £8.8 per week (£16.16) from its own resources, plus 4s. per day on behalf of the Commonwealth. This latter is a special extra benefit payable only to subscribers to the medical benefits organization. These two payments total £19.12, leaving a balance of £4.4 to be paid by Mrs. A. She will also receive an account from the hospital for operating room fees. There is no government subsidy for this but the medical benefits fund will repay £2.2 of this fee. The surgeon's fee will be about £17.17 for the operation and normal after-care. Mrs. A. will pay the account, which will state "Item 14 Appendicectomy." The receipted account will be sent to the medical benefits fund which will pay £9.7.6 from its own funds, and £5.12.6 on behalf of the government, totaling £15.15. The anesthetic fee will be similarly met on a basis of £1.17.6 from the fund and £1.2.6 from the government.

The public response to this scheme has been almost overwhelming, especially to the Medical Benefits Fund of Australia that is sponsored by the medical profession. The council of the British Medical Association has asked its members not to increase their fees until the scheme has been established for several years. This is to avoid political repercussions and embarrassment to the Liberal party in power that introduced the scheme. It will be remembered that the nationalized medical scheme which the Labor party endeavored to introduce in 1949 was a failure because of the refusal of the physicians to take part in it.

The Labor party today, therefore, is anxious to obtain political capital to discredit the present development, and that is why physicians are anxious to avoid any criticism of what they consider is the ideal solution to the problem of the cost of medical care.

No report of the Commonwealth Medical Benefits Scheme would be complete without a tribute to Sir Earle Page, the Minister for Health in the Commonwealth government, an outstanding surgeon, and a fellow of the Royal College of Surgeons, whose herculean efforts have been rewarded with the fruition of the final phase of this scheme of medical and hospital care.

ISRAEL

University Medical Library.—The circulating library for students opened at the Hebrew University-Hadassah Medical School some months ago contains 1,400 volumes, and additional gifts of books are frequently received. Most of the books have been presented by American-Jewish physicians, and a large collection has been sent by Dr. Louis Wolfson of Boston. The initiative in providing textbooks for the medical students was taken by the Phi Lambda Kappa medical fraternity of the United States, which is headed by Dr. Morton J. Robbins and Dr. M. M. Glazier. Many large American publishing firms have sent gift copies of their medical publications to the library. A collection of medical works presented by Dr. Walter Levy, a New Orleans surgeon, has been shipped to Israel by the Hadassah Chapter of that city, and part of this collection will be placed in the students' library. Sixty volumes on osteology have been presented by the Zionist Federation of South Africa. The opening of the lending library largely has solved the problem of providing medical students with necessary textbooks, which are very expensive.

American Students for the Medical School.—A limited number of students from the United States are to be admitted to the Hebrew University-Hadassah Medical School, according to a recent decision. The candidates must possess the same qualifications as are required by recognized American medical schools. The Jerusalem medical school will enroll candidates who have completed their premedical training in the United States, who have an adequate knowledge of Hebrew, and who have declared their intention of settling in Israel on completion of their studies.

Mass Immigration and Health Conditions.—In a review summarized in *Dapim Refuim*, May, 1953, Dr. Nassau reported on the "Mass Immigration and Health Conditions of the Old

Resident and New Population" in Israel. More than 200,000 children under 14, including 80,000 infants, have entered Israel during the great wave of immigration (1948-1952). The general health of the children of the resident population has not been affected by this immigration. No important damages of the general standard of nutrition have been observed. Among the children of the old residents, no deficiency diseases have been observed. The children of the new immigrants, on the other hand, suffered severe impairment, which was intensified because the health of some of them had already been damaged before or during their trip to Israel, as well as in the large immigrant camps. The rate of mortality at the pediatric wards increased threefold: 80 to 90% of the deceased children were new immigrants from the Middle East and Africa. The damages were in part due to (*a*) ignorance of proper child care and (*b*) the conditions under which the immigrants had to live, first in the immigrant camps and later in the transit camps.

The rate of childbirth is three times higher among immigrants from the Middle East than among the resident population. The Middle Eastern infants have no diseases due to the Rh factor, as the incidence of Rh-negative women from Yemen and Irak is very low (1 to 2%). The weight and body-length of these newborns is considerably less than of those in the resident population. The infant mortality rate rose among those of the transit camps in 1950 up to 15.7%; in 1951 it was still 8.2%, whereas the mortality in the cities was at the same period 3% and in the communal settlements 1.6%. The main causes of death among these infants were diseases originating from acute states of dystrophy. These dystrophies are again to be attributed to the ignorance of the mothers in questions of infant care and feeding. The infectious diseases that appeared most frequently at the transit camps were trachoma, favus, scabies, and infestation with lice. The children of the resident population were almost unaffected by these diseases. The number of new cases of schistosomiasis is (in spite of the great number of persons affected [20,000 to 30,000]) insignificant. A more serious problem, which also touches the children of the resident population, is the wide spread of worm infections.

Infectious diseases of the intestines such as typhoid, paratyphoid, and dysentery, affect almost exclusively new immigrants. Scarlet fever, acute hepatitis, and poliomyelitis were more frequently observed among the children of the old residents. There was a notable increase of diphtheria owing to the interruption of the vaccination during the period of the poliomyelitis epidemic. There are few cases of congenital syphilis. New cases of tuberculosis among the children of old residents and immigrants are rare. Only malaria, from which many of the newcomers are suffering, represents a latent danger to the resident population, though up to now there has been no increase in the number of new cases of malaria. Generally speaking, the mass immigration has not affected the general health of the resident population. The children of the newcomers have suffered severely owing to environmental conditions. Improvement of living conditions, teaching, and prophylaxis will make it possible to eliminate the still-existing dangers.

ITALY

Medical Days in Montecatini.—"Medical Days," organized by the management of the Montecatini Health Resort, were held in that city on May 16-17 for the sixth year. About 500 Italian internists and many guests from abroad were present. Prof. Cesare Frugoni, from the University of Rome and chief consulting physician of the resort, welcomed the guests and emphasized the importance that the Montecatini days are assuming in the cycle of Italian medical conventions. He then announced the topics to be discussed by two young Italian clinicians, Professor Cassano, director of the University of Pisa medical clinic, and Professor Coppo, director of the University of Modena medical clinic.

Since both topics dealt with factors that alter metabolism, Professor Frugoni pointed out the marked changes that modern life has produced in the diet of man, changes that disrupt his normal biochemistry and metabolism. To keep the metabolism normal, modern dietetics aims at a perfect balance between the caloric components (protein, lipids, and carbohydrates) and the noncaloric components (vitamins, water, and salts).

The numerous studies that have been made of the diet of the 2½ billion people of the world show that a large number of them have incongruous and poor diets; on the other hand, many persons eat more calories than they need, and they eat more food, especially lipid-containing foods, than they need.

Considering the damage that prolonged diet imbalances may produce, much of mankind is potentially ill from the standpoint of dietetics. Gillman's studies have clarified the ratio that exists between the frequency and the seriousness of liver alterations that occur in states of hyponutrition. The results of hypernutrition are just as severe, however, and among them are the steatosic alterations of the liver. These are today believed to play an essential part in development of cirrhosis of the liver that is due to disturbed nutrition.

Gastrocardiac Syndromes.—Professors Cassano, Tronchetti, and Bastieri discussed gastrocardiac syndromes. Professor Cassano said that every day it is common to observe some patients who complain of dyspeptic disturbances and who then are found to have heart disease. Cardiovascular disturbances that have their origin in the digestive system are common. The gastrocardiac syndromes seem to be closely related to a more general group of conditions related to the abdomen and the circulatory system, including syndromes involving the liver, gallbladder, and heart or the pancreas and the circulatory system. Careful examination of the morbid signs and diligent study of the physiopathological aspects, however, reveal that the gastrocardiac syndrome is quite distinct from the other syndromes involving the abdominal organs and the heart, even though there are many variants.

The gastrophrenic syndrome should not be classified as a gastrocardiac abnormality; it closely resembles the gastroanginal syndrome. Also the anginal syndrome caused by slight alimentary anaphylaxis should not be considered a gastrocardiac syndrome; the abdominal and cardiac distress in this illness is caused by hyposphyxia. The entire clinical picture, humoral and hemodynamic, can be reproduced by hypodermic injection of histamine in patients in whom liver disturbances resulted in an anaphylactic crisis with particular repercussions on the cardiovascular system.

A real gastrocardiac syndrome is present when a gastric alteration, that is, a true functional disturbance, can be demonstrated. The first manifestation of gastrocardiac abnormality is the hiatus syndrome in which an abdominal disturbance causes painful symptoms that can be related to the heart. The abnormally large hiatus and the consequent diaphragmatic hernia represent the abdominal source of conditions that affect the heart and the circulation.

Other gastrocardiac syndromes are closely related to aerophagy. The most typical aspect of this abnormality is the angor crisis of Roemheld's gastroanginal syndrome and its twin syndrome, paroxysmal tachycardia with gastric and respiratory complications (Rosenbach's syndrome). In both these syndromes there is a constant and typical critical change of the general hemodynamics, with fall of the systolic arterial pressure and especially the mean dynamic pressure and noticeable drop of the venous pressure.

The precordial sensation of constriction, the respiratory anguish, and the painful radiations create the sensation of angina pectoris in the patient. The physician has, therefore, the difficult task of recognizing the various aspects of the painful crisis and of distinguishing what is really anginal from what pertains to phrenic, gastric, and abdominal symptomatology. When the anginal nature of the pain has been recognized in the aerophagic patient, the functional character of these crises should be considered. Even though a favorable prognosis is made, it should always be remembered that distress, even functional distress, is always the expression of a phase of myocardial ischemia.

An animated discussion followed Professor Cassano's report. Professor Cassano emphasized that there is only one true angina. Then there are some painful thoracic syndromes, including the gastrophrenic syndromes. There are no false anginas. Lesions of the coronary arteries are common and may or may not become clinically apparent, depending on whether other neurovegetative and metabolic factors intervene.

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He proposes the classification of the obscure forms of angina under the term "minor angina," meaning by this a minor coronary entity with a better prognosis than true angina.

Noninfectious Liver Diseases.—Professor Coppo, a clinician from Modena, reported on the etiology of noninfectious liver diseases. His aim was to summarize the latest concepts of the mechanism of onset of many liver diseases. In many points the report represented the results of experiments and clinical observations of the speaker and his associates. In most cases the pathogenesis of liver disease is complex; this report dealt mainly with the metabolism-altering factors.

Discussing liver disease due to blood stasis, the speaker distinguished simple liver congestion, liver congestion with frank dysfunctional syndrome and often manifest jaundice, and cirrhosis or sclerosis of the liver.

Liver diseases that are due to bile stasis are often associated with infection of the biliary pathways. The pathogenetic mechanism of cholestatic liver disease is very complex. The speaker believes that, in addition to the inflammatory and mechanical factors, the repercussions produced on the digestive system by the absence of bile in the small intestine should be taken into consideration.

Liver diseases caused by altered protein metabolism are mainly due to lack of proteins. Altered metabolism of proteins and amino acids is an important pathogenetic factor of common cirrhosis. Hepatic alterations entirely similar to those produced by lack of proteins have been observed in patients with pellagra. Primary and secondary amyloid liver diseases are also included in this group.

Among the liver diseases associated with an altered carbohydrate metabolism, the speaker distinguished diabetic liver abnormalities from abnormalities due to glycogenesis (Gierke's disease). This is accompanied by surplus of liver glycogen that is produced by an altered metabolism.

Liver diseases associated with disturbances of fat metabolism assume various characteristics, depending on whether the steatosis and the accumulation of fats in the liver take place when there is a generalized lipidosis or when only the liver becomes filled with fats. In many cases, according to the speaker, liver steatosis constitutes one phase of the cirrhogenous evolution of the liver.

The speaker discussed also liver diseases associated with conditions caused by a deficiency of vitamins or hormones, those due to allergy and exogenous toxic factors, those of splenic origin, and those that develop during pregnancy. The hypothalamus seems to play an important role in liver diseases associated with nervous lesions; these are rare syndromes but they have great importance.

LONDON

Tuberculosis in Shoemakers.—A mass radiography survey of factory workers in Northamptonshire, the center of the shoemaking industry in Great Britain, revealed in 1947 a disturbingly high incidence of pulmonary tuberculosis in boot and shoe factories. Mortality figures indicated that the death rate for pulmonary tuberculosis was higher among "boot and shoe factory operatives" than among "makers and repairers" (nonfactory workers). These findings led to an intensive investigation, the results and implications of which are reviewed by Dr. Alice Stewart of the social medicine unit of the University of Oxford (*Brit. J. Tuberc.* **47**:122, 1953). The results can be summarized as follows: The relative excess of tuberculosis in the boot and shoe industry as a whole is due to the presence of an exceptionally large number of tuberculosis carriers in the working population. The unequal distribution of cases within the industry is due to the fact that in large workshops the spread of infection from carriers to fellow-workers is greater than in small ones. Since, for men, shoemaking is a relatively light occupation, it attracts more persons with established tuberculosis than most industries and allows them to remain at work much longer than if the work were really strenuous. Provided the men with chronic tuberculosis work on their own, as do most shoe repairers, no communal harm is done, but if, as is the case of boot and shoe factory workers, they work in large groups, the disease is spread to others. To account for the opposite trends of mortality among shoemakers and printers, it is "postulated that the migration of operatives from cottage workshops to factories, which is a feature of every industrial revolution, is liable to provoke a relatively slow rise in the tuberculosis death rate followed by a subsequent fall (i. e., a slow-motion epidemic). Provided, therefore, there is in any trade a nucleus of carriers, the date of its industrial revolution determines whether its tuberculosis death rate, relative to the national rate, is rising, falling or remaining stationary." In the shoemaking industry the factory system developed relatively late (1900-1914). From the data collected in this investigation it is suggested that, for every 10 boot and shoe operatives who contracted pulmonary tuberculosis from a relative, there were 18 to 20 who probably contracted it from a fellow-worker, and that for a shoemaker working in a room with about 200 other workers the risk of contracting tuberculosis is over three times as great as for a shoemaker working on his own.

Among the implications of this investigation, stress is laid on the fact that "spread of tuberculosis in factories is a greater menace to the community than spread in the home. . . . If the 'pressure head' of infection really lies in the factories, then our present policy of encouraging tuberculous subjects to return to industry as soon as they are fit is wrong and should be replaced by one in which tuberculous persons, while given every other consideration, are forbidden to mix with other factory workers. . . . In fact the only social remedy that could be relied upon to free the factories from infection would be 'a modified form of sanatorium treatment' for all tuberculous workers." Finally, it is stated that none of the studies described in this report "supports the view that shoemaking should be scheduled as a 'dangerous' occupation."

The Present Status of Cancer Research.—The present status of cancer research was recently reviewed by Prof. Alexander Haddow, director of the Chester Beatty Research Institute, Royal Cancer Hospital, in moving the adoption of the annual report at the 30th annual meeting of the British Empire Cancer Campaign. Until recently, little or nothing was known of the mode of action of carcinogenic agents. Lately, increased attention has been given to the biochemical action of these agents, and for the first time "in the case of certain cancer cells we have now recognized the imprint left upon them by the chemical agents which induced their appearance." The field of interest is being widened and now includes studies of the colloidal and molecular organization of the chromosomes. "Although we are not finally sure, it appears that the end result of such interference [i. e. with the chromosomes] may be a genetical and enzymatic deficiency—in other words, that the cancer cell is derived from the normal cell through the loss of enzyme or ferment systems normally responsible for growth regulation. While much remains to be done, I would hazard the prediction that sooner or later, depending on our efforts, we shall see in the chemotherapy of cancer something entirely new, namely a kind of substitution therapy by which the cancer cell will be controlled from without, by the same kind of chemical means in virtue of which the normal cell automatically regulates itself from within. While we cannot penetrate the future, the prospects have sensibly changed, and even cancer prevention, which only a few years ago seemed utterly beyond our hopes may yet prove to be within our ultimate powers."

He suggested that the history of cancer research may well resolve itself into three main phases: (1) the impartial exploration of every avenue of attack; (2) the definition and limitation of those few approaches that are decisive in strategy; and (3) a phase of conscious development. We may shortly be moving from the first of these phases to the second. "If the third phase is still beyond our ken, it may not elude us so very long. Although many biological paradoxes and puzzles have still to be solved, and many fantastic difficulties have yet to be overcome, the great fundamental principles, almost certainly of startling simplicity, are sure to emerge. It is then that we shall enter the phase of development." When this phase comes, "It will very likely partake of the nature of biological engineering on a vast scale, and may demand vast expenditures far in excess of those we have hitherto disposed of." His final

summing up was, "More than half-way through the twentieth century, after fifty years of cancer research in its modern sense, and after thirty years of concerted and ever-intensifying effort by the Campaign, it seems we are now at last approaching 'the end of the beginning'."

General Practitioners and Cancer Propaganda.—Earlier this year the British Empire Cancer Campaign sent a one-question questionnaire to every general practitioner in the United Kingdom: "Do you consider that the launching of a scheme of lay educational propaganda on cancer (by pamphlet, lectures, films, etc.) would be of assistance in securing the earlier diagnosis of cancer and thereby improving the chances of a cure?" To the 21,040 letters sent out, 5,053 replies were received, i. e., a 24% response. Of the replies, 2,148 were an unqualified "yes" and 2,683 an unqualified "no." In view of this strong element of objection among general practitioners to such propaganda, the campaign has decided to take no further action at the moment.

MEXICO

Tenth Anniversary of Children's Hospital in Mexico City.—In April, 1953, several functions commemorated the 10th anniversary of the Children's Hospital in Mexico City. In a public ceremony the founders of the hospital, former President Avila Camacho, former Secretary of Public Assistance, Dr. Baz, and former Undersecretary of Public Assistance, Dr. Zubirán, were honored. In the following days there were a pediatric meeting, a scientific exhibition, and a popular exhibition for medical education purposes.

At the pediatric meeting, which was attended by 458 physicians from all the states of Mexico and from abroad, there were general communications, roundtable discussions, and several seminars. The general communications presented the experience of the hospital in its first 10 years of life. Among the subjects discussed, the nutritional problems, the infectious diarrhea of children, and the acute abdominal conditions of infancy aroused the most interest. The scientific exhibition, which was also presented by the staff of the hospital, had 45 booths, each dedicated to illustration of the problems that are of most importance to pediatricians and general practitioners.

The popular exhibit was visited daily by an average of 12,000 persons. There were 60 booths, in many of which special lecturers explained to the general public different aspects of the care of children. Many booklets were distributed without charge.

Acute Abdominal Conditions in Infantile Diarrhea.—At the meeting on pediatrics, commemorating the 10th anniversary of the Children's Hospital, Drs. Carlos Sariñana, Joaquín de la Torre, and Arturo Fonseca, from the departments of surgery and medicine, reported their experience with 33 patients under 2 years of age who had an acute abdominal condition following an attack of diarrhea. Twenty-three (69.5%) were under one year of age, the youngest being only 13 days old. The diarrhea was considered severe in 79% of the cases, and in 76% blood was found in the stools; it appeared during the second week in almost half of the cases.

In every case there was abdominal distention and vomiting. In only one-third of the cases was intense spasm found, and in 80% constipation appeared together with the acute abdominal symptoms. The most frequent complication was bronchopneumonia, which was present in 45% of the cases. Stool cultures were done on 20 patients, with positive results in 11. Organisms of the colon and paracolon group were isolated in six, including coli 3 Kauffman in one, Shigella flexneri in two, Proteus morganii in one, Pseudomona aeruginosa in one, and Salmonella typhi and Endamoeba histolytica in one. Roentgenograms of the abdomen showed dilated loops, fluid levels, and thickened intestinal walls. In cases of perforation free air was found in the subdiaphragmatic space.

Twenty-four of the patients were operated on, with 22 deaths. All of the nine patients not operated upon died. Treatment consisted of use of antibiotics, correction of fluid and electrolyte balance, and administration of blood and plasma.

Autopsy was done in 15 cases, and in 10 more the resected small intestine was studied histologically. In the rest, diagnosis was based on the operative findings. Different types of lesions that caused the acute abdominal symptoms were found at the time of laparotomy or autopsy. These were classified by the authors as follows: 1. Echymotic or necrotic lesions, with circumscribed zones of infarction or pinpoint hemorrhages. The most frequent site was the ileum, followed by the jejunum or colon. In only one case were lesions found in the stomach and duodenum. Some areas were perforated, with evident peritonitis. 2. Extensive zones of intestinal infarction, usually located in the ileum. Thrombosis of the mesenteric vessels proved to be the cause of the infarct in two of these patients. 3. Ulcerations of the small or large bowel of variable depth, and perforation secondary to such ulcerations. 4. Zones of intestinal pneumatosis, usually located in the ileum. 5. Bloody or purulent fluid in the abdominal cavity, found in all of the patients.

The pathology department reported the following findings in the intestine: 1. Edema, congestion, necrotic areas, and in some cases hemorrhages of the mucosa and submucosa. In others, the mucosa had disappeared completely and was replaced by granulation tissue. 2. Round cell or plasma cell infiltration of one or several layers. Sometimes edema and hemorrhage of the serosa and subserosa, with deposits of fibrin and leukocytes, were found. In others, there was complete autolysis with perforation. Peritonitis and mesenteric adenitis were described, as well as alterations in other organs. This, together with the results of the bacteriological studies showing different micro-organisms, led to the conclusion that, in some cases, peritonitis was a manifestation of a septicemia of intestinal origin.

The most important comments referred to the differential diagnosis between truly acute conditions of the abdomen and conditions due to paralytic ileus. The etiology and pathology of the disease are not completely understood, but the authors believe that infection, malnutrition, and increased intra-abdominal pressure are contributing factors in the majority of cases. The surgical procedure most frequently used was the resection of the affected part, with enterostomy.

Medical Meeting at San Luis Potosí.—At the central city of San Luis Potosí, from April 28 to May 2 this year, a regional medical meeting was attended by physicians from all the neighboring states and from Mexico City. Medical meetings like this, called "cycles of medical days," have been held since 1940 and the last one was the fifth. They are sponsored by the University of the State of San Luis Potosí and the Central Hospital and the Society for Medical Studies of the city of San Luis Potosí.

The committee that organized this meeting consisted of Dr. J. M. Torre, Dr. J. E. Sanchez, and Dr. E. Anaya, as president, secretary, and treasurer respectively; all of them are members of the faculty of the university and of the staff of the Central Hospital.

The meeting consisted of (*a*) general communications and symposiums, (*b*) clinicopathological conferences, (*c*) special sections, and (*d*) medical motion pictures. The general communications were presented by members of the major hospitals of Mexico City. They concerned surgery of the mitral valve, by Drs. Chavez, Robles, and Mendoza, from the Instituto de Cardiología; portal hypertension, by Drs. Sepulveda, Villalobos, Rojas, Falomir, Muñoz, and Quijano from the Hospital de Enfermedades de la Nutrición; tumors of children, by Drs. Salas and Contreras from the Hospital Infantil; and endocrine problems in gynecology, by Drs. Alvarez-Bravo, Arzac, and Gómez-Mont.

The section meetings were on endocrinology, cardiology, cancerology, and anesthesiology. Thirty-seven papers were read in all. The clinicopathological reports were presented by members of the local hospital and discussed by the guests. In addition there were clinical rounds, surgical demonstrations, and exhibits of different kinds. The general consensus was that this meeting was very successful and that the comparison of the experience of the local physicians with that of physicians from other cities was very useful.

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CORRESPONDENCE

EXPEDITION TO SOUTH AMERICA

To the Editor:—I would like to place before your readers details of a project that might interest them. An expedition has been planned for late this fall. The members of the expedition will drive two sports cars, towing small trailers, down the Pan-American Highway to Rio de Janeiro. We hope to complete the journey entirely under the power of the cars, and, as many readers of THE JOURNAL may know, this is a pioneering venture.

There are three of us at present, an American who is a graduate of Massachusetts Institute of Technology, with motor engineering experience, and two Englishmen who both are Oxford graduates, with some years in the United States, and one who has a master's degree in business administration from Harvard. We need a fourth man to complete the group. As the fourth man we want to recruit a young American medical graduate, preferably with some experience in the tropics. Expenses will be shared but will be low, since the expedition is receiving financial support in return for a photographic record of the journey. Time will be available for study en route so this expedition might be an opportunity that some reader would like to hear about. Further details may be obtained by writing to P. O. Box 1610, Grand Central Station, New York 17.

BRIAN ACWORTH
Inter-American Expedition
27 Grosvenor Place
London, S.W.1, England

OBSTRUCTION DUE TO SERUTAN

To the Editor:—Several cases have been reported in which esophageal obstruction resulted from the use of one of the hygroscopic bulk laxatives. In every instance some of the ingested material lodged in the esophagus and formed a tenacious, sticky mass. An additional case seems worth describing, not only as a further warning concerning the use of such laxatives but, particularly, as a report of the prompt response to a method of treatment that, as far as I know, has not previously been tried.

A 72-year-old white man was admitted to the North Carolina Baptist Hospital at 4 p. m. on April 7, 1953, because of an obstruction in the esophagus. For years he had been in the habit of taking Serutan. The manufacturer of Serutan states that it is a hydrogel obtained from Plantago ovata and extracted from sugar beets from which the roughage has been removed. At 10 p. m. on the night before admission the man put 2 teaspoonfuls of Serutan in a glass of water and swallowed the contents of the glass before allowing the powder to dissolve. In a few minutes, he experienced a painful, burning sensation when he attempted to swallow and found that he could not drink a glass of water. Throughout the night he vomited and coughed up what he described as "thick clots of mucus." A physician who was called at 6:30 a. m. gave him an intramuscular injection of 130 mg. (2 grains) of sodium phenobarbital in oil. This made him drowsy, but did not relieve the obstruction. Physical examination at the time of admission was noncontributory. Roentgen examination was not attempted because of the possibility that the barium might add to the obstruction. Since Serutan is a carbohydrate material, it was thought that a starch digestant might dissolve the mass; hence the patient was given 8 cc. of liquid Taka-diastase in half a glass of water, to be sipped slowly. Before the first dose had been completed, the patient said that he felt "the lump melting away" and found that he could swallow without effort. The next morning (April 8) he was able to take a bland meal without difficulty, and he was discharged that afternoon. He made an uninterrupted recovery.

A fluoroscopic examination of the esophagus, with a barium swallow, was made a week later, on April 14. The examiner reported "no obstruction to the flow of barium through the esophagus. . . . There was on deep inspiration a very minimal degree of hiatus insufficiency."

The rapid relief obtained from the liquid Taka-diastase, after more than 20 hours of obstructive symptoms, justifies the supposition that it was effective in liquefying the obstructing mass. Certainly it seems to be worthy of further trial in cases in which esophageal obstruction follows the ingestion of a hygroscopic laxative. Taka-diastase should be used before barium is given since the barium would add to the size of the mass in the esophagus and would also act as a mechanical barrier between the starchy material and the starch digestant.

WINGATE M. JOHNSON, M.D.
Bowman Gray School of Medicine
of Wake Forest College
Winston-Salem 7, N. C.

REMOVAL OF SKIN SUTURES

To the Editor:—I enjoyed the article on removal of skin sutures by Weinstein and Roberts in THE JOURNAL, July 25, 1953, page 1231. Dr. Daniel L. Borden introduced a similar traction loop into the suture which he reported in the *American Journal of Surgery* (**73**:100, 1947). I have added one more step. I leave the end of the interrupted sutures long and tie the long ends together, or I use a split buckshot in place of the knot so that all stitches can be removed at once. This saves digging around for each stitch and makes it less likely that a stitch will be missed because the light is poor at the bedside, tissue swelling is present, or the area is inaccessible.

R. H. KAZMIERSKI, M.D.
208 Oak St.
Coudersport, Pa.

To the Editor:—After reading the article "Easy Removal of Skin Sutures," by Drs. Weinstein and Roberts (*J. A. M. A.* [July 25] 1953) we agree with the authors that their suture lift is a valuable method for removing skin sutures. We have found, however, that it is easier to use the eye end of the needle in inserting the lift. This blunt end slips under the stitch easily without piercing the skin. We also tie the loose end of the first knot to the loose end of the last knot so that the suture lift can be easily identified. With these two additions to the technique, we have found the time necessary for insertion of the lift markedly reduced.

CONRAD A. ETZEL, M.D.
R. WALLACE JOURNEY, M.D.
Chester Hospital
9th and Barclay Sts.
Chester, Pa.

To the Editor:—In THE JOURNAL for July 25, 1953, page 1231, Weinstein and Roberts describe an easy method of removal of skin sutures. A practically identical method was described by A. P. Hovnanian in *Plastic and Reconstructive Surgery* (**8**:407 [Nov.] 1951). I have used this method myself during the past year and a half and find it very satisfactory.

ROBERT H. IVY, M.D.
Editor, *Plastic and Reconstructive Surgery*
1930 Chestnut St.
Philadelphia 3.

To the Editor:—In reference to "Easy Removal of Skin Sutures" (*J. A. M. A.* **152**:1231 [July 25] 1953), a very similar method for skin suture removal was described in 1951. The reference is "A Simple Technic for Removal of Fine Interrupted Skin Sutures" (Hovnanian, A. P.: *Plast. & Reconstruct. Surg.* **8**:407, 1951).

PAUL J. SCHNEIDER, M.D.
2930 McClure St.
Oakland 9, Calif.

COUNCIL ON MEDICAL SERVICE

RATING COMMUNITIES

During the past two summers the Council's Committee on Extension of Hospitals and Other Facilities [1] has been making a study of physicians placement activities in a number of states. In these studies the Committee arranged for field visits to various small communities in each state, some of which had obtained a physician recently and others of which had requested a physician but had been unable to attract one. In communities that had been successful in obtaining a physician, questions were asked of the physician and his wife designed to learn why he had located there rather than in some other community in the state. In communities where a physician had not been obtained, an effort was made to learn why the community had been unsuccessful in its efforts to attract a physician.

As a result of the first series of field visits, a list was compiled showing (*a*) the major factors related to a community's need for a physician and (*b*) the primary characteristics determining a community's attractiveness to a physician. The various categories were then assigned a relative importance numerically and an A, B, C scale set up to record point totals. From this it was theoretically possible, on the basis of adequate knowledge, to assign a community an A, B, or C priority rating that would relate to its need for physician as well as its ability to attract one.

This medical priority rating program was then ready for testing in the field. This was done in a second series of field visits to small communities. After talks with physicians in these communities, particularly those who had recently established their practices, and after interviews with community leaders in those places that were still searching for a physician, the data gathered were applied to the priority program and a rating assigned to each community. This rating was then checked against the opinion of the executive secretary of a state medical society or some other qualified person familiar with the medical needs and resources of that community. In nearly every case the evaluation given by one with firsthand knowledge concerning the location coincided with the rating shown by the priority plan.

By means of such testing several revisions were made in the plan. The revised categories and the points assigned to them were reviewed by the Committee on Extension of Hospitals and Other Facilities and the Council on Medical Service and the priority rating program as approved by them is published here for review by physicians and others interested in placement problems.

A MEDICAL PRIORITY RATING PROGRAM

This Medical Priority Rating Program has been prepared and is submitted to the state medical associations with full knowledge of the many differences between states. However, the criteria on which a physician bases his appraisal of the desirability and medical need of a community are sufficiently uniform to permit their adaptation to any state. In such adaptation, the points assigned to items may be increased or decreased to conform to local conditions without altering the basic concept.

Encouraging the state associations to use this program in the operation of their placement service is admittedly an experimental step. At the same time it may well serve several purposes: (1) to stimulate communities to analyze their characteristics and facilities from a physician's viewpoint; (2) to encourage state associations in the collection of a certain minimum amount of information on each opening, and (3) to supply the A. M. A. with a valuable differentiation among the communities in the United States listed as needing a physician.

1. The Committee members are Ralph A. Johnson, M.D., Chairman, Detroit; Elmer Hess, M.D., Erie, Pa.; Walter E. Vest, M.D., Huntington, W. Va.; E. Dwight Barnett, M.D., New York; John W. Cline, M.D., San Francisco; Cleon A. Nafe, M.D., Indianapolis, and Willard A. Wright, M.D., Williston, N. D.

Objectives

I. To determine where a need for a physician actually exists and estimate the degree of that need

II. To evaluate the medical desirability of a given location

III. To assist communities in improving their medical desirability rating

IV. To guide physicians in their choice of locations

Need Priority—From the Standpoint of the Community

A.....60 to 105 points
B.....30 to 60 points
C..... 0 to 30 points

Calculation To Be Based on:

1. Doctor-population ratio
   - 1 active to 1,200 population and under.... 0 points
   - 1 active to 1,200 to 2,000 population.....20 points
   - No M.D. or 1 active to 2,000 population and over .....30 points
2. Distance to nearest adequate medical care
   - Within trade area..... 0 points
   - Inadequate, or not within trade area.....25 points
   - (Considered inadequate if 20 or more points are recorded for number 1)
3. Age of physicians serving the area
   - Under 45 years of age..... 0 points
   - 45 to 60 years of age.....10 points
   - No M.D., one over 60 years of age, or one in limited practice.....35 points
   - (Example: Doctor #1 50 years..10 points; Doctor #2 33 years.. 0 points; Average..... 5 points)
4. Intangible factors .....0 to 15 points
   - A. Need as seen by the inhabitants of the community
   - B. Their willingness to accept the situation
   - C. Any factors which may increase or decrease the need picture as drawn by the above categories

Desirability Priority—From the Standpoint of Physician

A.....70 to 105 points
B.....40 to 70 points
C.....under 40 points

Calculations To Be Based on:

1. Hospital facilities
   - Located in community.....30 points
   - Reasonably accessible to physicians.....15 points
   - Not reasonably accessible..... 0 points
2. Office facilities offered by the community
   - Adequate space and equipment offered at low rent or rent free for a given period.....25 points
   - Adequate space, no equipment, with rent....20 points
   - No facilities or inadequate ones..... 0 points
3. Opportunity for assured income as health officer, care of the indigent, industrial accounts, etc.
   - Yes .....15 points
   - No ..... 0 points
4. Facilities for family life.....0 to 25 points
   - If excellent, allow.....25 points
   - If good, allow.....15 points
   - If poor, allow..... 0 points
   - A. Housing .....0 to 25 points
   - B. Schools .....0 to 25 points
   - C. Churches .....0 to 25 points
   - D. Social opportunities, including golf clubs, fraternal organizations, other professional men in the community, women's organizations .....0 to 25 points
   - A + B + C + D ÷ 4..Average:——
5. Intangible factors .....10 points
   - A. Availability of diagnostic service, consultation and postgraduate education
   - B. Ambulance and nursing service available
   - C. Possibility of time off for vacations and special causes
   - D. Proximity to a large medical center
   - E. Other factors contributing to medical desirability

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MEDICAL LITERATURE ABSTRACTS

UNITED STATES

A.M.A. Arch. Ind. Hyg. & Occupat. Med., Chicago

7:455-546 (June) 1953

Effect of Modifications of Surface of Quartz on Its Fibrogenic Properties in Lungs of Rats: 1. Quartz Leached with Ringer's Solution. 2. Quartz Etched with Hydrofluoric Acid. 3. Quartz Coated with Coal Extract. E. J. King, G. P. Mohanty, C. V. Harrison and G. Nagelschmidt. —p. 455.

Acute Toxicity of Mineral Dusts. J. C. Dale and E. J. King.—p. 478.

Adsorption of Dyes, Amino Acids, Proteins, and Metal Hydroxides on Quartz. J. C. Dale and E. J. King.—p. 484.

Field Equipment for Collection and Evaluation of Toxic and Radioactive Contaminants. W. B. Harris, H. D. LeVine and M. Eisenbud. —p. 490.

Spectrochemical Analysis of Beryllium in Biological Tissue. J. Y. Ellenburg and L. E. Owen.—p. 503.

Radon Inhalation Studies in Rats. S. H. Cohn, R. K. Skow and J. K. Gong.—p. 508.

Hyperthermic and Pathologic Effects of Electromagnetic Radiation (350 Mc.). J. E. Boysen.—p. 516.

Acute Narcotic Effects of Monochloromonobromomethane Vapor in Rats. C. C. Comstock, R. W. Fogleman and F. W. Oberst.—p. 526.

Control of Environment in Prevention of Industrial Accidents. B. H. Harvey.—p. 529.

A.M.A. Arch. Internal Medicine, Chicago

91:695-824 (June) 1953

Insulin Reactions: Manifestations and Need for Recognition of Long-Acting Insulin Reactions. R. K. Maddock and L. P. Krall.—p. 695.

Syndrome of Myelofibrosis: Report of Eight Cases. J. E. Cook, J. W. Franklin, H. E. Hamilton and W. M. Fowler.—p. 704.

Clinical Method for Measuring Capillary Blood Pressure and Its Application in Hypertension. E. Davis.—p. 715.

Tolerance to Nasogastric Tube Feedings: Comparative Clinical Study of Two Dietary Formulas. E. B. Smith, E. E. Wollaeger and S. M. Victor. —p. 721.

*Needle Liver Biopsy in Thyrotoxicosis. E. R. Movitt, B. Gerstl and A. E. Davis.—p. 729.

Clinical Studies in Geriatrics: I. Serum Lipid Partitions. J. Pomeranze, L. J. Boyd and A. A. Goldbloom.—p. 740.

*Idiopathic Ulcerative Colitis: Giant Ulceration Following Corticotropin (ACTH) and Cortisone Therapy. E. C. Texter Jr., C. W. Legerton Jr., J. M. Ruffin and others.—p. 744.

Diagnostic Importance of Café-au-Lait Spot in Neurofibromatosis. F. W. Crowe and W. J. Schull.—p. 758.

Studies on the Aging Heart: The "Normal" Heart in Old Age—Study Based on Analysis of 800 Autopsies. H. Poliakoff and P. Kaufman. —p. 707.

Treatment of Central Nervous System Cryptococcosis: Laboratory Studies. C. A. Carton and C. S. Liebig.—p. 773.

Successful Corticotropin (ACTH) Therapy of Whipple's Disease Refractory to Cortisone. K. Plummer, B. B. Wesiger III and C. M. Caravati. —p. 784.

*Vitamin E and Climacteric Syndrome: Failure of Effective Control as Measured by Menopausal Index. M. H. G. Blatt, H. Wiesbader and H. S. Kupperman.—p. 792.

Needle Liver Biopsy in Thyrotoxicosis.—Biopsy specimens of the liver were obtained with the Vim-Silverman needle in 13 patients with thyrotoxicosis. The anatomical material thus obtained was correlated with the results of hepatic function tests, including the determination of the serum bilirubin, serum gamma globulin, serum proteins with albumin-globulin ratio, cephalin cholesterol flocculation, thymol turbidity, sulfobromophthalein dye excretion, and plasma prothrombin. The authors found that the functional capacity of the liver in thyrotoxicosis is hardly affected or is affected only to a very slight extent and that pathological changes in the liver in this condition are slight except for vacuolization of liver cell nuclei. They believe that the discrepancy between their own observations and those of other investigators, who observed severe anatomical changes in the liver of patients with thyrotoxicosis is due to the fact that the previous authors made their observations on autopsy material exclusively.

Ulcerative Colitis: Giant Ulceration Following Corticoid Therapy.—These observations were made on 14 patients with a severe or fulminating ulcerative colitis who were treated with intravenous administration of corticotropin at the Duke Hospital and on one other patient treated elsewhere. The clinical course of the disease prior to the treatment was classified as fulminating in 11 and as severe in four. Fourteen patients received daily intravenous infusions of 20 units of corticotropin for approximately 12-hour periods for from 4 to 17 days. Twenty courses were given to the 14 patients. Three patients also received corticotropin intramuscularly (80 units per day) for periods of 5 to 13 days, and one patient was treated with corticotropin gel (40 to 80 mg. per day) for a 16 day period prior to intravenous therapy. Cortisone was taken by four of the patients either before or after the course of corticotropin. Antibiotics were administered concurrently. Sustained improvement resulted in eight of the patients subjected to corticoid therapy, while the remaining seven either had only temporary improvement or became worse. Perforation of the colon developed in one patient, and large undermined ulcers developed in four patients who subsequently underwent colectomy. The colectomy specimens showed longitudinal undermined ulcers unaccompanied by significant fibrosis or mucosal regeneration. The sigmoidoscopic appearance of these ulcers and the accompanying roentgenologic changes were distinctly different from what is usually seen in cases of idiopathic ulcerative colitis in which corticoid therapy is not given. Corticotropin and cortisone appear to alter the course of idiopathic ulcerative colitis. Since submission of this paper the authors followed an additional patient, who was observed to have huge linear ulcers, one of which perforated, transecting the entire length of the transverse colon, following intravenous therapy with corticotropin. At autopsy the anterior abdominal wall was adherent to a large open segment of the transverse colon, 27 cm. in length and 5 cm. in width. The anterior wall of the transverse colon appeared to be completely destroyed by the ulcerative process. There was also a localized abscess associated with perforation of the cecum and ascending colon. The remaining colon showed huge, linear ulcers, surrounded by considerable mononuclear inflammatory reaction.

Vitamin E and the Climacteric Syndrome.—Blatt and associates attempted to evaluate the effectiveness of alpha tocopherol therapy in women with menopausal symptoms with the aid of the menopausal index, which gives numerical values to the commonest menopausal symptoms. While in an earlier report the authors employed a menopausal index including 11 symptoms, they now consider only the four most important symptoms, namely, vasomotor symptoms, nervousness, insomnia, and paresthesia. The effect of vitamin E was compared to the effect of two standard estrogens, ethinyl estradiol and conjugated estrogenic substances (equine). In addition, phenobarbital and placebos were used throughout the study, which extended over three years. Vitamin E administered in the form of 50 to 100 mg. of alpha tocopherol acetate a day was no more active than a placebo in the control of menopausal symptoms and proved to be effective in only 25.6% of 82 patients. Placebo therapy proved effective in 33% of 298 patients. Phenobarbital was therapeutically successful in 42.8% of 88 climacteric patients. Ethinyl estradiol, 0.05 mg. a day, and conjugated estrogenic substances (equine), 1.25

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Titles marked with an asterisk (*) are abstracted.

mg. a day, were comparable in activity and adequately controlled menopausal symptoms in 92.6% and 90.8% of 107 and 173 patients respectively. This failure of vitamin E to exert a therapeutic effect corresponds with its failure to show pharmacological activity experimentally in the absence of vitamin E deficiency.

A.M.A. Arch. Ophthalmology, Chicago

49:591-714 (June) 1953

Geniculocalcarine Injuries in War Casualties. N. S. Jaffe and L. S. Durkin.—p. 591.
Bilateral Endophthalmitis Phacoanaphylactica: Pathologic Study of Lesion in Eye First Involved and, in One Instance, the Secondarily Implicated, or "Sympathizing," Eye. J. A. deVeer.—p. 607.
Local Scleral Necrosis: Late Sequel of Irradiation. I. S. Jones and A. B. Reese.—p. 633.
Perimetry with Ultraviolet (Black) Radiation and Luminescent Test Objects: Preliminary Report. D. O. Harrington.—p. 637.
Opnthalmoplegic Migraine: Pathogenesis: Report of Pathological Findings in Case of Recurrent Oculomotor Paralysis. D. O. Harrington and M. Flocks.—p. 643.
Functional Evaluation of Pupillary Light Reflex Pathways: Experimental Pupillographic Studies in Cats. O. Lowenstein, S. B. Murphy and I. E. Loewenfeld.—p. 656.
Fibrinoid Degeneration of Tarsal Conjunctiva. W. Kornblueth, A. Laufer, E. Loebel and M. Frisher.—p. 671.
Diseases of Retina. A. E. Maumenee.—p. 675.

A.M.A. Arch. Pathology, Chicago

55:443-530 (June) 1953

*Lesions of Pancreas in Malignant Hypertension: Review of 100 Cases at Necropsy. G. T. Hranilovich and A. H. Baggenstoss.—p. 443.
Pathology of Mumps Virus Meningoencephalitis in Mice and Hamsters. J. R. Overman, J. H. Peers and L. Kilham.—p. 457.
Experimental Induction of Iron Overload in Rat: I. Morphological Alterations Due to Dietary Siderosis. J. P. Wyatt and J. Howell. —p. 466.
Vascularity of Early Subcutaneous Nodule of Rheumatoid Arthritis. L. Sokoloff, R. T. McCluskey and J. J. Bunim.—p. 475.
Relation of Glycogen, Phosphorylase, and Ground Substance to Calcification of Bone. J. D. Cobb.—p. 496.
Effect of High Pyridoxine Intake in Cholesterol-Fed Chicks. W. McFarland.—p. 503.
Effects of Anterior Hypophysis on Mammary Glands and Adrenals. R. Silberberg, M. Silberberg and M. Opdyke.—p. 506.
Mechanism of Softening of Tubercles: II. Behavior of Desoxyribonuclease in Tubercles Developing in Lungs of Rabbits. C. Weiss and F. M. Singer.—p. 516.

Lesions of Pancreas in Malignant Hypertension.—Hranilovich and Baggenstoss of the Mayo Clinic say that in a previous study of dilatation of pancreatic acini in patients who died of uremia, they were impressed by the high incidence of other parenchymal lesions in cases in which uremia was the result of nephrosclerosis. Since the parenchymal lesions in cases of nephrosclerosis were usually associated with a severe degree of arteriolosclerosis, it was surmised that most of the parenchymal lesions were the result of vascular changes and could be interpreted as infarcts. This study endeavors to elucidate the relationship between the vascular changes and the various parenchymal lesions. Records of 100 cases of malignant hypertension observed between 1924 and 1948 in which necropsies were performed were obtained from the files of the Mayo Clinic. The clinical charts were abstracted for data referable to age, sex, onset, duration and degree of hypertension; symptoms referable to cerebrospinal, cardiovascular, and renal systems; alterations in chemical constituents of blood and other body fluids, and type of death. The necropsy protocols furnished data concerning weights of the hearts and kidneys and changes in other organs. Gross descriptions of the pancreas were studied in every case. The routine sections of the pancreas were reviewed. The controls consisted of 100 cases in which the sex and the age distribution by decades of life were essentially the same as those in the cases of malignant hypertension. Clinical charts, correspondence, and necropsy protocols in the control cases were used to rule out hypertension and congenital or acquired cardiac disease. Moderate to severe arteriolosclerosis occurred in 68, infarcts of the pancreas were found in 7 cases, focal parenchymal necrosis in 21 cases, and foci of atrophy in 73 of the 100 cases of malignant hypertension. Vascular alterations were the most important factor in the production of parenchymal lesions such as infarction, focal parenchymal necrosis, atrophy, and fibrosis. Although arterial thrombosis could be demonstrated in 6 of the 7 cases of pancreatic infarction, it could not be demonstrated in any of the 21 cases in which parenchymal necrosis was observed. It is probable that congestive cardiac failure and shock may be important factors in the production of parenchymal necrosis in the presence of a severe arteriolosclerosis. Arteriolosclerosis of the pancreas is strongly indicative of, but not pathognomonic for, malignant hypertension. Vascular factors by themselves are not important etiological factors in acute hemorrhagic pancreatitis.

American Heart Journal, St. Louis

45:795-948 (June) 1953

Excretion of Sodium-Retaining Substances in Patients with Congestive Heart Failure. B. Singer and J. Wener.—p. 795.
Effect of Edema and Dietary Sodium on Efficacy of Sodium Removal by Cation Exchange Resin. L. E. Duncan Jr.—p. 802.
Determination of Oxygenation of Blood in Vitro by Using Reflected Light. F. A. Rodrigo.—p. 809.
Techniques for Facilitating Quantitative Analysis of Spatial Vectorcardiograms. G. H. Whipple.—p. 823.
Simplified Method for Determining Angle Between Two Spatial Vectors. R. A. Helm and N. O. Fowler Jr.—p. 835.
Spatial Vector Analysis of Early Right Ventricular Preponderance. J. C. Dahl and E. Simonson.—p. 841.
Effect of Lithium Chloride on Electrocardiogram of Isolated Perfused Heart. W. A. Butcher, K. G. Wakim, H. E. Essex and H. B. Burchell. —p. 855.
Further Observations on "Sustained" Hypertension in Rat. S. M. Friedman, M. Nakashima and C. L. Friedman.—p. 864.
*Functional Evaluation of an Internal Mammary Coronary Artery Anastomosis. A. Vineberg and D. Miller.—p. 873.
Valvular Stenosis as Cause of Death in Surgically Treated Coarctation of Aorta. G. Jacobson, R. S. Cosby, G. C. Griffith and B. W. Meyer. —p. 889.
Ventricular Standstill During Intravenous Procaine Amide Treatment of Ventricular Tachycardia. M. A. Epstein.—p. 898.
Action of New Oral Preparation of Digitalis, Acetyldigoxin. C. D. Enselberg, B. N. Brodoff and S. I. Griboff.—p. 909.
*New Device for Objective Auscultation of Heart: Permanent Audible and Visual Record of Heart Acoustics. A. Briskier.—p. 914.
Heart Scarab of Ancient Egyptians. S. Rodbard.—p. 918.
Congenital Aortic Atresia and Hypoplasia of Aortic Orifice: Case Reports on Two Members of a Family. V. G. Brekke.—p. 925.
Fatal Reaction to 1-Hydrazinophthalazine (Apresoline). B. M. Cohen —p. 931.

Internal Mammary Coronary Artery Anastomosis.—The anatomical structure of the ventricular myocardium and the pathological conditions existing in coronary heart disease suggested to Vineberg and Miller that an implanted internal mammary artery can bridge the blocked superficial coronary vessels which are still open and free of disease. A new approach to the vascularization of the myocardium was first attempted by them in 1945. As a result of experiments on several series of dogs they concluded that coronary artery disease is limited to the epicardial vessels. The vessels within the myocardium are, for the most part, not diseased. There exists, therefore, within the myocardium, a vast network of patent vessels capable of carrying arterial blood to the myocardium. Fresh arterial blood reaches this network when an internal mammary artery is placed with an open sixth intercostal artery between the myocardial fibers. The authors feel that the estimation of the value of experiments designed to revascularize the myocardium should be based on functional studies. In their experiments they demonstrated that the internal mammary artery after implantation into the myocardium is capable of maintaining the circulation of the left ventricle after complete or partial occlusion of the anterior descending branch of the left coronary artery. These experiments suggested that internal mammary artery implantation might be of value in the treatment of human coronary artery insufficiency. To date nine patients with coronary artery insufficiency have undergone internal mammary artery implantation. The results have been most encouraging and will be published.

New Device for Objective Auscultation of Heart.—In order to register simultaneously the heart acoustics for visual and audible reproduction, three known instruments (eventually to be combined into one unit) are used: the electrocardiograph, the phonostethograph, and the electromagnetic tape recorder. While the limb and chest leads pick up the electrical waves of the heart beats and transmit the magnified waves upon a mov-

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ing medium, the microphone simultaneously picks up the heart sounds and murmurs. These are first carried to the same moving medium for visual recording of the sound, which is the known electrostethogram; the same sounds and murmurs picked up by the microphone are simultaneously recorded on a sound magnetic tape. This simultaneous visual and aural recording of the heart beats, sounds, and murmurs can be called an audiovisocardiogram. This permanent record can be studied, compared, and discussed at any time. It can be kept on file or shipped anywhere to be examined by a physician who did not examine the patient. The sounds are less distorted, the tape giving higher fidelity than the phonographic disk. The permanent visual and audible record will enable the physician to follow a given disease step by step. In many instances, questionable murmurs become clarified. Any eventual change in the quality of a sound or murmur can be added and compared with those previously recorded. The audiovisocardiogram will reduce the margin of errors so often encountered in auscultation and may prove of value in a variety of medical fields: in rheumatic mitral stenosis, in the surgical treatment of congenital heart disease, and during labor (changes in fetal heart beats could be recorded). The method might prove useful for teaching purposes, in clinics, in induction centers, factories, insurance companies, and in legal medicine.

Am. Journal of Clinical Pathology, Ann Arbor, Mich.

23:519-622 (June) 1953

Symposium on Clinical Hemoglobinometry. F. W. Sunderman, R. P. MacFate, D. A. MacFadyen and others.—p. 519.

Indirect Colorimetric Determination of Cholesterol. T. V. Feichtmeir and J. Bergerman.—p. 599.

Normal and Abnormal Values of Protein-Bound Iodine: Obtained by Chloric Acid Method. B. Zak, A. M. Koen and A. J. Boyle.—p. 603.

New Test for Bilirubin in Urine. H. Sobotka, A. V. Luisada-Opper and M. Reiner.—p. 607.

Tablet Test for Bilirubin in Urine. A. S. Giordano and M. Winstead. —p. 610.

Use of Sequestrene as Anticoagulant. G. G. Hadley and N. L. Larson. —p. 613.

Rapid Identification of Pasteurella Pestis Using Specific Bacteriophage Lyophilized on Strips of Filter Paper: Preliminary Report. D. C. Cavanaugh and S. F. Quan.—p. 619.

American J. Digestive Diseases, Fort Wayne, Ind.

20:149-185 (June) 1953

Hematologic Studies in Atrophic Gastritis. M. E. Dailey, M. J. Ellenhorn and E. H. Falconer.—p. 149.

Massive Resection of Small Intestine: Six Year Follow-Up Study. J. K. Berman, E. D. Habegger and E. Billings.—p. 152.

Compatible Hyperglycemia. A. Sindoni Jr., P. Gerber, F. Bove and L. Zibold.—p. 157.

Iron Equilibrium: Clinical Observations on Nutritional Anemia. A. C. Johnson.—p. 179.

American Journal of Medicine, New York

14:651-782 (June) 1953

Effect of Weight Reduction and Caloric Balance on Serum Lipoprotein and Cholesterol Levels. W. J. Walker, E. Y. Lawry, D. E. Love and others.—p. 654.

*Hypercholesterolemia, Myxedema and Atherosclerosis. H. L. Blumgart, A. S. Freedberg and G. S. Kurland.—p. 665.

Failure of Ingestion of Cream to Affect Blood Coagulation. J. A. Tulloch, R. S. Overman and I. S. Wright.—p. 674.

*Evaluation of Anticoagulant Therapy in Myocardial Infarction Based on Prognostic Categories. R. H. Furman, C. O. T. Ball, R. G. Gale and others.—p. 681.

Control of Prothrombin Activity with Tromexan Therapy. M. Weiner, G. Simson, J. J. Burns and others.—p. 689.

Clinical Evaluation of Ethyl Biscoumacetate (Tromexan). J. B. Vander Veer, E. H. Funk Jr., F. R. Boyer and E. A. Keller.—p. 694.

Clinical Use of New Anticoagulant, Phenylindanedione: Report of 400 Cases. A. Blaustein, N. Shnayerson and R. Wallach.—p. 704.

Serum Cholinesterase in Health and Disease. L. J. Vorhaus and R. M. Kark.—p. 707.

Evaluation of Anticoagulant Therapy. I. S. Wright.—p. 720.

Hypercholesterolemia, Myxedema, and Atherosclerosis.—Clinical and postmortem findings are reported in five men and three women between the ages of 22 and 63 with rheumatic heart disease or cor pulmonale. They had all survived one to 13 years following total surgical thyroidectomy and had clinical hypothyroidism or myxedema with increased cholesterol values. These patients were generally maintained with basal metabolic rates of approximately −20% by small doses of thyroid. The blood cholesterol levels of these patients averaged 125 mg. above the average pretreatment levels of 170 mg. per 100 cc. Cholesterol values of over 400 mg. per 100 cc were present for many months in some of these patients. Postmortem examinations did not reveal complete occlusion of any of the coronary arteries of these eight patients. Five of the eight showed no narrowing of any of the main stems or branches of the coronary arteries. Only three of the eight showed slight narrowing of one of the main stems. In the other arteries atheromatosis and atherosclerosis varied greatly but were similar to that generally observed in similar euthyroid patients. The authors' observations do not disprove the role of the cholesterol in the production of atherosclerosis. They demonstrate, however, that the hypothyroid state with its hypercholesterolemia is not necessarily and by itself a sufficient etiological factor for the production of coronary atherosclerosis.

Anticoagulant Therapy in Myocardial Infarction.—Bishydroxycoumarin (Dicumarol) alone or bishydroxycoumarin and heparin were given to 100 patients during episodes of acute myocardial infarction. An earlier group of 211 patients receiving conventional treatment but no anticoagulants served as controls. Eighteen of the anticoagulant-treated patients died (a mortality rate of 18%), while 68 of the control group died (a mortality rate of 32%). Patients were classified in prognostic categories according to the following criteria: (1) very poor risks, in whom the attack was the patient's first and shock and diabetes or shock and azotemia were present, or who had had a previous attack and in whom shock was present or congestive failure and diabetes and azotemia were present; (2) poor risks, in whom shock, congestive failure, diabetes, or azotemia was present within the first week of admission; and (3) good risks, in whom none of the concomitant manifestations in group 1 and 2 were present. Anticoagulants failed to improve the mortality rate in the "very poor risk" category. The mortality rate in the "good risk" category was very small with or without anticoagulants. The beneficial effects of anticoagulants were most apparent in the "poor risk" group of patients and then chiefly in those less severely ill. Such beneficial effects cannot be entirely ascribed to a reduction in thromboembolic phenomena, since thromboembolic complications in the authors' experience played a minor role in the course of acute myocardial infarction. The lower mortality rate in these patients treated with anticoagulants was probably due either to some property of these agents in addition to their anticoagulant property or to other entirely unrelated and undetected factors.

American Journal of Physiology, Washington

173:189-378 (May) 1953. **Partial Index**

Effects of ACTH, Cortisone and Hydrocortisone on Renal Functions of Hypophysectomized Dogs. D. P. Earle, S. J. Farber, R. C. de Bodo and others.—p. 189.

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Metabolic Effects of Testosterone Propionate in Experimental Diabetes. P. M. Wright and C. D. Kochakian.—p. 217.

Removal of Radioactive Gold Colloid by Perfused Mammalian Liver. R. C. Little and H. B. Kelly.—p. 265.

Size of Largest Arterio-Venous Vessels in Various Organs. D. B. Gordon, J. Flasher and D. R. Drury.—p. 275.

Comparison of Epinephrine and ACTH Action on Circulating Eosinophils of Mice. R. S. Speirs and J. Sullivan.—p. 282.

Uptake of P by Cardiac Muscle in Vivo. J. B. Marsh, G. G. Casten and H. F. Elliott.—p. 297.

Some Effects of Dihydrogenated Ergot Alkaloids on Renal Hemodynamics, Water and Electrolyte Excretion in Dog. W. D. Blake. —p. 337.

Influence of Renal Electrolyte Composition on Tubular Excretion of P-Aminohippurate. J. V. Taggart, L. Silverman and E. M. Trayner. —p. 345.

Effect of X-Ray to Kidney on Renal Function of Dog. M. L. Mendelsohn and E. Caceres.—p. 351.

Pantothenic Acid Deficiency in Experimental Renal Hypertension in Dogs. L. A. Lewis and I. H. Page.—p. 359.

Hemolytic Anemia of Dog Following Bilateral Nephrectomy. E. E. Muirhead, F. Jones, J. A. Stirman and W. Lesch.—p. 371.

Am. J. Roentgenol. & Rad. Therapy, Springfield, Ill.

69:907-1080 (June) 1953

Dissecting Aneurysms of Thoracic and Abdominal Aorta: Report of Six Cases, with Discussion of Roentgenologic Findings and Pathologic Changes. G. S. Lodwick.—p. 907.
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Variation of Diaphyseal Development which Simulates Roentgen Appearance of Primary Neoplasms of Bone. D. H. Allen.—p. 940.
*Intrauterine Perforation of Meckel's Diverticulum. S. Rosza and R. J. Gross.—p. 944.
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*Treatment of Moderately Advanced Carcinoma of Prostate with Radioactive Gold. H. D. Kerr, R. H. Flocks, H. B. Elkins and D. Culp. —p. 969.
Simplified Intracavitary Injection Technique for Radioactive Gold. J. U. Hidalgo, S. B. Nadler and R. T. Nieset.—p. 978.
Circulation of Radiation Damaged Skin: Radiosodium Clearance Studies. B. Roswit, L. H. Wisham and J. Sorrentino.—p. 980.
Grid Depth Dose Investigation for 200 and 400 Kilovolts at Center and Edge of Field. L. E. Jacobson.—p. 991.
Effect of Selective Shielding of Rabbit Intestine During Total Body Irradiation. R. S. Farr and P. H. De Bruyn.—p. 1001.

Intrauterine Perforation of Meckel's Diverticulum.—The case reported is of interest primarily because one of the complications of Meckel's diverticulum, ulceration and perforation, occurred during gestation and caused the appearance of roentgen findings commonly associated with meconium ileus. Only subsequent postmortem examination, nine hours after birth, disclosed the true nature of the roentgen findings. The specimen of the small intestine showed a diverticulum measuring 3.5 cm. in length and 2.5 cm. in diameter in the ileum approximately 3 cm. from the ileocecal junction. The diverticulum was ruptured and the gap covered over by thickened peritoneum, which itself was the site of a marked inflammatory reaction and had a thick greenish-brown fibrinopurulent exudate on its surface. The walls of the diverticulum were thin, necrotic and also covered with this patchy exudate. The cut surface of the intestine revealed a hemorrhagic, necrotic wall. The mucosa in the diverticulum was largely sloughed. A review of the world literature disclosed no similar case.

Radioactive Gold in Treatment of Carcinoma of Prostate.—Older methods of irradiation proved unsatisfactory in cancer of the prostate, because, although sufficient radiation could be applied to destroy the tumor, adjacent normal structures, notably the rectum and bladder, received too much radiation. With the advent of Au^{198}, whose main radiation component is beta rays, the prostate seemed a site in which to try this isotope. The first patient was a 72-year-old man who had had a transurethral resection for obstruction, and had been treated with diethylstilbestrol for the five months after it had been demonstrated that cancer of the prostate was present. Radioactive gold was used, and in three weeks the large, hard mass was impalpable. Ninety-two days after the injection, biopsy specimens were negative for carcinoma. In the past 17 months, the authors treated 120 cases of inoperable carcinoma of the prostate with radioactive gold. All possible operative procedures are carried out before the injection of the gold is begun. After estimation of the amount of tissue involved by malignancy, the radiologist measures the amount of radioactivity desired and dilutes it to the proper volume. He then loads the protected syringe and passes it to the urologist who does the injecting. Usually from 8 to 16 cc. of the diluted material of Au^{198} are injected into the prostate from within the bladder. The periprostatic region and the seminal vesicles are injected transvesically and extravesically. Palpable nodes are also injected. If the estimated amount of involved tissue was between 20 and 35 gm., the authors used 3 mc. per gram; if 35 to 70 gm., 2 mc. per gram; if 75 to 150 gm., 1 mc. per gram of tissue. Because of complications, this dosage schedule was later reduced to 1 mc. per gram; but the regression of the tumors so treated has not been as satisfactory as with the earlier dose. At the present the authors feel that a moderate sized prostate, between 30 and 60 gm., is probably best treated with a millicurie per gram ratio of 2 to 1. About 98% of the patients treated showed rapid decrease in the size of the palpable neoplasm. Eleven of 37 cases with postinjection biopsies showed no evidence of neoplasm. Of the 50 patients observed from 6 to 17 months, 27 are alive without evidence of clinical disease and seven died without evidence of carcinoma. Eight are alive with clinical disease and eight died with the disease. Complications are almost exclusively confined to rectal irritation. There were 15 cases in the first 50 and three in the subsequent 67 with this complication. Four developed rectal ulcers, three of which required colostomy.

Am. Practitioner & Digest of Treatment, Philadelphia

4:305-374 (May) 1953

Medical Management of Poliomyelitis Problem: Part II. R. Batson. —p. 305.
Subacute Bacterial Endocarditis. G. A. Vance.—p. 312.
Emotional States with Special Reference to Cardiovascular System. A. Bernstein.—p. 321.
Aneurysms: Their Evaluation and Treatment. J. W. Lord Jr.—p. 326.
Hydrogenated Ergot Alkaloids (Hydergine): A Hypotensive Agent. F. B. Schultz.—p. 330.
Neurological and Neurosurgical Approaches to Cerebral Palsy. P. Robb. —p. 337.

4:375-430 (June) 1953

Therapy of Acute Leukemia. K. C. Kelty and M. F. Beard.—p. 375.
Treatment of Symptomatic Leucorrhea with New Preparation. J. Rock and J. M. Nauss.—p. 382.
Pregnancy in the Diabetic. A. Weinstein.—p. 384.
Phenylbutazone (Butazolidin®) in Musculoskeletal Diseases, Bronchial Asthma and Other Allergic Diseases. A. Rowe Jr., R. W. Tufft, P. G. Mechanick and A. H. Rowe.—p. 390.

American Review of Tuberculosis, New York

67:707-894 (June) 1953

Tuberculosis Today and Tomorrow. D. T. Smith.—p. 707.
Method for Evaluating Attitudes of Tuberculous Patients. G. Calden. —p. 722.
Observations on Effect of Induced Hyperglycemia on Glucose Content of Cerebrospinal Fluid in Patients with Tuberculous Meningitis. J. E. Sifontes, R. D. B. Williams, E. M. Lincoln and H. Clemens.—p. 732.
Pulmonary Function in Pregnancy: II. Comparison of Effects of Pneumoperitoneum and Pregnancy in Young Women with Functionally Normal Lungs and Serial Observations During Pregnancy and Postpartum Pneumoperitoneum. W. E. Patton, W. H. Abelman, N. R. Frank and others.—p. 755.
Id.: III. Serial Observations in Patients with Pulmonary Insufficiency. E. A. Gaensler, W. E. Patton, J. M. Verstraeten and T. L. Badger. —p. 779.
Toxicity of Isoniazid for Rhesus Monkey. L. H. Schmidt, R. Hoffman and H. B. Hughes.—p. 798.
*In Vitro and In Vivo Activity of Streptomycin and Isoniazid Singly and in Combination. G. L. Hobby, T. F. Lenert, Z. C. Rivoire and others. —p. 808.
Toxic Lipid Component of Tubercle Bacillus ("Cord Factor"): II. Occurrence in Chloroform Extracts of Young and Older Bacterial Cultures. H. Noll and H. Bloch.—p. 828.
Id.: III. Occurrence and Distribution in Various Bacterial Extracts. J. Asselineau, H. Bloch and E. Lederer.—p. 853.
*Caseation Necrosis in Human Brucellosis: Report of Case. J. B. Crow, D. M. Tormey, W. J. Redner Jr. and B. H. Sullivan.—p. 859.

Action of Streptomycin and Isoniazid Singly and in Combination.—Hobby and associates present the following results of in vitro and in vivo studies that indicate the high degree of antituberculous activity of isoniazid against both streptomycin-susceptible and streptomycin-resistant organisms. The emergence of resistance to combinations of streptomycin and isoniazid occurs more slowly than to either compound alone. Neither compound is capable of preventing the ultimate emergence of resistance to the other. Cross-resistance between streptomycin and isoniazid may occur under certain conditions despite the fact that isoniazid is effective against streptomycin-resistant organisms not previously in contact with isoniazid and that streptomycin is effective against isoniazid-resistant organisms not previously in contact with streptomycin. It was concluded, therefore, that streptomycin and isoniazid, when used under appropriate conditions and in adequate quantities, may act synergistically both in vitro and in vivo. Not enough data are available to permit the assumption that the impressions gained from the observations reported would apply to all strains of Mycobacterium tuberculosis. There is some

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evidence that the action of isoniazid alone and in combination with streptomycin against Myco. butyricum may differ from its action against the H37Rv strain of Myco. tuberculosis. Further evidence is needed to confirm this impression and to prove whether or not all strains of Myco. tuberculosis var. hominis parallel the strains used in the present study.

Caseation Necrosis in Human Brucellosis.—A 21-year-old man was hospitalized on May 23, 1951, with the chief complaints of vomiting and diarrhea. He had been in good health until May 21, 1951. He was aware of an asymptomatic generalized lymphadenopathy for several weeks prior to this illness. For almost a year preceding the illness he had been employed in a meat-packing plant where he slaughtered cattle and hogs. Some three months before this hospitalization, he sustained a laceration of his lower lip when struck by the thrashings of a pig. This lesion healed. His temperature now was 102.2 F, and he was acutely ill. Several nontender firm cervical lymph nodes, 0.5 to 2 cm. in size, were palpable. Similar lymph nodes could be felt in the supraclavicular, axillary, inguinal, and epitrochlear regions. Brucella suis recovered by blood cultures was highly susceptible in vitro to chloramphenicol, aureomycin, and streptomycin and was resistant to penicillin and sulfadiazine. A supraclavicular lymph node was biopsied on June 1, 1951, and on pathological examination it was found to consist of five firm, gray-white lymph nodes with yellow, granular cut surfaces. Microscopically, the lymph nodes were poorly circumscribed and partially encapsulated. Almost the entire section was composed of large confluent masses of avascular caseation necrosis. Scattered foci of calcification were noted. Because of the clinical evidence of toxicity and abnormal urine findings, the patient was given 1.5 gm. of aureomycin daily for 12 days, or a total of 18 gm. There was prompt clinical improvement characterized by subsidence of fever, malaise, nausea, vomiting, diarrhea, and abdominal distress. The lymphadenopathy noticeably regressed. The Brucella agglutination titer gradually rose, and blood cultures five weeks after the onset of the acute illness yielded the growth of Brucella suis. A second course of aureomycin was instituted consisting of 0.5 gm. every six hours for 14 days, or a total of 28 gm. Blood cultures became negative following this therapy, and there was a gradual fall of the agglutination titer. The patient regained 10 lb. (4.5 kg.) in weight. On discharge, he was asymptomatic and afebrile, but two blood cultures after return from a convalescent furlough were positive for Brucella suis, and so the patient was recalled for further therapy. A review of the literature of pathological material from the Armed Forces Institute of Pathology indicates that the inflammatory reaction to the Brucella organism is, in general, nonspecific.

American Surgeon, Atlanta, Ga.

19:491-584 (June) 1953

Neurogenic Tumors of Anterior and Middle Mediastinum: Report of Two Cases. L. E. Tebow and R. B. Brown.—p. 491.
Fourth Dimension in Pulmonary Disease. R. H. Overholt and S. W. Atwell.—p. 498.
Cholelithiasis and Cardiac Disease. R. M. Garrett.—p. 503.
Retrograde Jejuno-Gastric Intussusception: Review of Literature and Report of Case. P. Lavadia Jr., B. W. Haynes Jr. and M. E. De Bakey.—p. 507.
Repair of Neglected Ruptured and Severed Achilles Tendon. W. J. Tobin. —p. 514.
Recurrent Carcinoma of Ampulla of Vater: Case Report. J. P. Woodhall and M. Mass.—p. 523.
Management of Postoperative Pulmonary Bronchial and Vascular Congestion. W. S. Edwards.—p. 528.
Clinical Evaluation of Succinylcholine (Win 7907): Report of Its Use in 100 Cases. O. A. Zeller Jr., J. W. McGowan and W. F. Sergent. —p. 533.
Surgical Treatment of Stenotic Valvular Disease. W. J. Burdette.—p 540.
Endometriosis. J. B. Lukins and L. Lukins.—p. 562.
Congenital Esophageal Atresia with Tracheo-Esophageal Fistula: Report of Five Cases. G. B. Hodge and G. D. Johnson.—p. 569.
*Herniation of Lumbar Intervertebral Disks in Coal Miners: Preliminary Report. E. L. Gage and W. A. Shafer.—p. 577.

Herniation of Lumbar Intervertebral Disks.—Gage and Shafer have operated since 1946 on 564 patients for herniation of intervertebral disks. The report deals with 303 of these patients, who were coal miners. Some of these men worked at loading coal while on their knees or in a stooped position in coal tunnels no higher than 26 to 40 in. (65 to 100 cm.). Direct injury was claimed by 35.9% as a cause of their complaints but in only 22.7% was a connection with work injuries accepted. A rather high percentage of the miners (nearly two-thirds) had multiple disk involvement. This may be due to the fact that many with single disk lesions may have become symptom-free with conservative treatment. No disk lesion was found in 3.6% at operation. Plain roentgenograms and laboratory tests were not sufficient for diagnosis, which was made primarily by a neurological examination supplemented in 14% of the cases by myelography. At the last follow-up 277 of the miners either returned to work or were convalescing satisfactorily. Their ability to remain at work must be determined by a five year follow-up study. The authors believe that miners with true disk herniation who have not been relieved by conservative treatment should be operated upon and that the majority of these patients can then return to work at the mines. Most miners who have had multiple disk lesions should not work in extremely low coal seams, however.

Blood, New York

8:491-586 (June) 1953

Concentration of Intrinsic Factor and Vitamin B12-Binding Activities of Fractions of Desiccated Hog Stomach. W. H. Krusoff, A. D. Welch, R. W. Heinle and G. C. Meacham.—p. 491.
*Some Observations on Anemia in Rheumatoid Arthritis. M. R. Jeffrey. —p. 502.
Erythrophagocytosis in Case of Plasma Cell Leukemia. C. E. Butterworth Jr., W. B. Frommeyer Jr. and W. H. Riser Jr.—p. 519.
Nature of Neutral Red Staining Refractive Particles in Lymphocytes of Blood of Normal Humans. L. H. Hempelmann and N. P. Knowlton Jr.—p. 524.
Effect of Horse Serum, Adrenal Hormones, and Histamine on Number of Eosinophils in Blood and Peritoneal Fluid of Mice. H. Panzenhagen and R. Speirs.—p. 536.
*Control of Postirradiation Hemorrhagic State by Platelet Transfusions. M. C. Woods, F. N. Gamble, J. Furth and R. R. Bigelow.—p. 545.
Proteolytic and Fibrinolytic Activity of Serum: Activation by Streptokinase and Staphylokinase Indicating Dissimilarity of Enzymes. E. E. Cliffton and D. A. Cannamela.—p. 554.
Preservation of Leukocytes. J. L. Tullis.—p. 563.
Use of Nitrogen Mustard in Management of Two Pregnant Lymphoma Patients. K. W. Deuschle and W. S. Wiggins.—p. 576.

Anemia in Rheumatoid Arthritis.—Moderate anemia is a common feature of rheumatoid disease but its origins remain obscure and its treatment is frequently unsatisfactory. The presence of anemia may hinder adequate physical or orthopedic treatment and may produce a degree of lassitude that considerably aggravates the joint disability. Investigations on the following aspects of this anemia are reported: (1) changes in the peripheral blood and bone marrow, (2) blood, plasma, and corpuscle volumes, and (3) changes in iron metabolism. The patients studied had active rheumatoid arthritis, uncomplicated by any disease liable to affect hemopoiesis. The patients were partly "selected" by the fact that this hospital receives a predominance of cases refractory to outpatient treatment, such as gold or simple physiotherapy. The series included patients of various ages, differing widely in the severity and duration of their disease, and is believed to represent a fair sample of cases of active rheumatoid arthritis associated with anemia. The anemia was essentially normocytic and hypochromic, the mean corpuscular volume being usually normal and the mean corpuscular hemoglobin content and concentration usually reduced. Anemia was more pronounced in the presence of very active disease. The myelogram suggested impaired maturation of normoblasts and retarded hemoglobin formation. The blood and plasma volumes were not essentially abnormal, though the frequent asthenic habitus and loss of weight might produce apparently exalted values in terms of body weight. Changes common to many anemias were found in the relatively severe cases of anemia, that is, normal plasma volume with decreased corpuscle and whole blood volume. The plasma iron concentration was usually reduced; subnormal values were invariable in cases with very active disease or marked anemia. The iron-binding capacity of the serum might be raised by iron deficiency or slightly reduced but not enough to be a factor in producing or maintaining anemia. As judged by changes in the plasma iron, intestinal absorption of iron after a single maximal therapeutic dose was variable. Some cases

showed the excellent absorption and therapeutic response characteristic of simple iron deficiency. Others gave evidence of impaired absorption.

Platelet Transfusion in Postirradiation Hemorrhagic State.—The hemorrhagic state following massive irradiation is characterized by the appearance of erythrocytes in large numbers in the lymph. The peak of this erythrocyte diversion into the lymph is reached in rats at 9 to 14 days and in dogs at 11 to 17 days after irradiation. The parallel between the postirradiation hemorrhagic tendency and the decrease in circulating platelets has been confirmed by several workers. A new impetus to studies of the role of platelet deficiency in radiation injury was given by the work of Cronkite and coworkers, who demonstrated that daily injections of platelet suspensions begun before thrombocytopenia developed prevented the hemorrhagic phase. This study describes the extent to which platelet injections diminish the hemorrhagic tendency of irradiated dogs and rats as indicated by erythrocyte counts in the lymph. Suspensions of concentrated, homologous blood platelets when introduced intravenously into lymph-cannulated dogs and rats (9 to 12 days after exposure to an LD_{50} or greater dose of x-rays) eliminated the large scale diversion of erythrocytes into the lymph during the hemorrhagic phase of radiation injury. By means of injection of platelets preinjection levels of below 50,000 were raised to an average of 107,000 in 6 dogs, and to an average of 310,000 in five rats at 15 to 30 minutes after injection. All platelet-injected animals showed clearing of the bloody lymph within the first hour after injection, and within five to six hours the average erythrocyte count in the lymph of dogs dropped from an average of 270,000 to an average of 12,000 and in the rats from an average of 900,000 to an average of 30,000. No depression of lymph erythrocyte counts was found in control rats receiving plasma only. Lymph erythrocyte counts in the platelet recipients remained low for 24 to 36 hours after injection; bleeding into the lymph space reappeared in both rats and dogs when the platelet level dropped below approximately 50,000. Blood platelet and lymph erythrocyte levels are excellent indexes of the severity of the hemorrhagic phase of radiation injury and for gaging the results of therapeutic procedures. While the mechanism of platelet action remains to be demonstrated, the present studies support the thesis of earlier workers that platelets are essential to maintain vascular integrity and that a platelet deficiency is the main cause of the postirradiation hemorrhagic state.

California Medicine, San Francisco

78:487-532 (June) 1953

Corticotropin (ACTH) and Cortisone: Newer Concepts of Their Use in Clinical Practice. L. W. Kinsell, L. Boling, J. W. Partridge and N. Foreman.—p. 487.
Organic Phosphorus Poisoning in General Practice: Parathion, TEPP, HEPT, EPN and Others. P. H. Leach.—p. 491.
Chest Injuries Among Korean Casualties. H. J. Chapman, E. H. Dickinson, E. Rollins and others.—p. 496.
Endoscopy in Hernia at Esophageal Hiatus. S. J. Stempien and A. D. Silk.—p. 499.
Diseases and Deaths: Summary of Data from Los Angeles County General Hospital, 1918 to 1948. E. Bogen, E. M. Butt and A. Djang.—p. 501.
Practical Device for Leg Traction at Home. A. M. May and G. F. Norman.—p. 505.
Testicular Torsion and Acute Epididymitis: Procaine Infiltration of Spermatic Cord as Aid in Differentiation. G. I. Smith.—p. 506.
New Vasoconstrictor—Preliminary Report. H. Steinberg.—p. 507.
*Pneumoperitoneum in Perforated Peptic Ulcer: Factors in Roentgenographic Demonstration. W. Gaines.—p. 508.
Pyloroplasty for Gastric Drainage with Vagotomy. F. B. Wilkins.—p. 513.

Roentgenographic Demonstration of Pneumoperitoneum in Perforated Peptic Ulcer.—The proved cases of perforated ulceration of the stomach and duodenum observed at the San Francisco City and County Hospital in the eight year period ended July 1, 1950, were reviewed. All cases in which there was a final diagnosis of perforation of benign ulceration were included, whether or not the perforation was sealed off or adherent to the liver or the pancreas at the time of operation or autopsy. The total was 337 cases. Roentgenologic examination was performed in 324 cases. A review of the data on these 324 cases indicated that pneumoperitoneum is more likely to be demonstrated in roentgen films made within six hours after perforation than in films made after a longer interval. In several cases films made early did not show intraperitoneal gas and later films did. There appeared to be no constant relationship between the size of the perforation and the incidence of demonstrable pneumoperitoneum; it was less often demonstrated in cases of posterior perforation than when the lesion was at an anterior site. In many cases roentgen examination was performed with the patient in both the erect and the left lateral decubitus positions. These two roentgenographic views were equally reliable, and there was agreement between them in 94% of the cases. Clinical factors may influence the decision as to which position should be used. It is easier for a patient with acute pain in the abdomen to turn on his side than to sit erect. Also, the lateral decubitus position may facilitate the escape of intraluminal gas if there is considerable fluid in the stomach. If the physical condition of the patient permits, it would seem wise to take films in both positions.

Cancer Research, Chicago

13:397-476 (June) 1953

*Mechanisms Responsible for Origin and Distribution of Blood-Borne Tumor Metastases: Review. D. R. Coman.—p. 397.
Effect of Hypophysectomy on Development of Adrenal Tumors in C3H Mice. D. J. Ferguson and M. B. Visscher.—p. 405.
Studies on Origin of Naturally Occurring Antibodies Against Tumor Viruses Developing in Aging Chickens. F. Duran-Reynals, B. R. Burmester, G. E. Cottral and E. Bryan.—p. 408.
Studies on Metabolism of 2-Benzoylaminofluorence-9-C^{14} and 2-Acetylaminofluorine-9-C^{14} in Rat. H. R. Gutmann and J. H. Peters.—p. 415.
Transplantation of Human Brain Tumors to Brains of Laboratory Animals. H. S. N. Greene.—p. 422.
Promoting Action of Croton Oil in Skin Tumorigenesis. M. Klein.—p. 427.
Effect of Various Compounds on Ehrlich Ascites Carcinoma. K. Sugiura.—p. 431.
Failure of Biotin or Oxybiotin to Exert a "Procarcinogenic" Effect on Tumor Formation by 4-Dimethylaminoazobenzene. A. E. Axelrod and K. Hofmann.—p. 442.
*Hypercalcemia, a Complicaton of Hormone Therapy of Advanced Breast Cancer. B. J. Kennedy, D. M. Tibbetts, I. T. Nathanson and J. C. Aub.—p. 445.
Genesis and Growth of Tumors. VI. Effects of Varying Level of Minerals in Diet. A. Tannenbaum and H. Silverstone.—p. 460.
Studies on Intracellular Composition of Livers from Rats Fed 2-Acetylaminofluorene. A. K. Laird and E. C. Miller.—p. 464.

Blood-Borne Tumor Metastases.—This paper reviews concepts of the mechanisms of metastasis in the light of knowledge gained from experiments during the past 10 or more years. The local invasiveness of cancer cells is primarily dependent upon loss of the mutual adhesiveness of the cells, which loss is associated with, if not due to, local calcium deficiency. The easily detached single cancer cells, or small clusters of them, are ameboid and are thereby enabled to penetrate the adjacent tissues. Distant dissemination of cancer cells is largely dependent upon entrance of cells into vascular channels through which they are carried to other parts of the body. Duration of growth of the primary tumor bears a positive relationship to the number of metastases, whereas the size of the primary tumor is less effective in influencing the number of metastases. The venous distribution of metastases is largely dependent upon the anatomical arrangement of the venous channels (including the vertebral veins) into which emboli are commonly released. This accounts for the frequency of metastatic involvement of the lungs, the liver, and the bones of the spine, pelvis, and skull. The arterial distribution of metastases is also dependent upon the number of embolic cells reaching the various organs, and in addition depends upon whether the embolic cancer cells lodge in capillaries and sinusoids or in thick-walled arterioles. These phenomena are variable from organ to organ, and may account for the relative infrequency of metastasis to some organs (spleen, muscle) and the frequency of metastasis to other organs (adrenals, kidney). The lungs, although frequently the site of metastatic tumors, apparently afford relatively adverse conditions for tumor growth. This may be attributable to the peculiar physical structure. Single embolic cells or tiny clusters, rather than large clumps, give rise to most metastases, because these cells are able to reach the capillary bed whereas

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large clumps lodge in arterioles. "Soil" factors affecting the distribution of metastases seem to consist chiefly of physical or mechanical circulatory differences rather than of chemically favorable or unfavorable environments for the growth of tumor cells.

Hypercalcemia, a Complication of Hormone Therapy in Breast Cancer.—Kennedy and associates call attention to the fact that hypercalcemia is a potential serious complication during androgenic and estrogenic hormone therapy of patients with advanced breast cancer. Nine patients with obvious hypercalcemia were culled from a series of 361 women with advanced cancer of the breast who were treated with sex steroid hormones. The complication occurred only in patients with osseous involvement. Eighty-four of 253 patients treated with estrogens and 97 of 108 treated with androgenic hormones demonstrated osseous metastases. Of these, seven patients treated with androgens and two patients treated with estrogens developed manifestations of the hypercalcemia syndrome. Two patients with hypercalcemia, not included in the above consecutive series, were studied recently and have been incorporated in this report. The case histories of the 11 patients are presented. Metabolic studies of patients with "induced hypercalcemia" demonstrated an elevated serum calcium, hypercalcuria, altered serum and urinary phosphorus levels and occasionally renal insufficiency. Immobilization, possible acceleration of tumor growth, and removal of barriers to the spread of cancer cells may contribute to the development of the hypercalcemia. The resemblance to the metabolic effects of parathyroid hormone is described in one case. The occurrence of hypercalcemia as a complication of hormone therapy of breast cancer does not contraindicate its general use. Nor does the presence of a spontaneous elevation of serum calcium preclude hormone therapy. Once hypercalcemia is manifest, discontinuance of the hormone may be followed by a decrease of the serum calcium. Parenteral administration of fluid to prevent severe dehydration and measures to correct the electrolyte imbalances are helpful. A low calcium diet aids the reduction of the serum calcium. Sodium citrate may be given to reduce temporarily the amount of ionized calcium in the blood by formation of a soluble, nonionized calcium citrate complex. Caution must be exercised in regard to the total amount of sodium citrate given in a day, because if the renal function is impaired, alkalosis may be produced.

Diseases of Chest, Chicago

23:595-712 (June) 1953

Pulmonary Function and Circulatory Dynamics in Artificial Pneumoperitoneum: I. Studies on Patients with Pneumoperitoneum Therapy for Pulmonary Tuberculosis. R. C. Kory, D. C. Roehm, G. R. Meneely and R. A. Goodwin Jr.—p. 595.

Id.: II. Studies on Patients with Pneumoperitoneum as Therapeutic Measure in Pulmonary Emphysema. R. C. Kory, D. C. Roehm, G. R. Meneely and R. A. Goodwin Jr.—p. 608.

*Significance of Cell Types in Bronchogenic Carcinoma. H. J. Moersch and J. R. McDonald.—p. 621.

*Pulmonary "Coin" Lesions. H. G. Trimble.—p. 634.

Hypersensitivity Reactions to Oral Para-Aminosalicylic Acid. R. H. Hayes and M. Weiss.—p. 645.

*Kartagener's Syndrome in Children. L. B. Dickey.—p. 657.

Adrenal Function in Chronic Pulmonary Tuberculosis. J. W. Goldzieher and J. S. Edlin.—p. 667.

Cavernous Metastatic Pulmonary Carcinoma: Report of Two Cases. E. Salzman, J. H. Reid and G. I. Ogura.—p. 678.

Viability and Chemotherapeutic Sensitivity of Tubercle Bacilli Isolated from Pulmonary and Other Lesions in Embalmed Bodies at Autopsy. Case Report and Clinico-Pathologic Correlation of Cases Studied to Date. B. H. Johnson, F. Davis, R. W. Jamison and W. J. Marti.—p. 686.

Histoplasmin Sensitivity in Brazil. J. Silveira.—p. 693.

Cell Types in Bronchogenic Carcinoma.—The records of 1,000 patients with proved carcinoma of lung, taken at random from the files of the Mayo Clinic, were reviewed. Of the 895 men and 105 women between the ages of 22 and 80 years, 90 had small cell carcinoma, 137 had adenocarcinoma, 378 had large cell carcinoma, and 395 had squamous cell carcinoma. The ratio of men to women was 29:1 with small cell carcinoma and 25.3:1 with squamous cell carcinoma. The difference in the ratio of men to women was not so great in the two other cell types, being 3.4:1 for adenocarcinoma and slightly less than 6:1 for large cell carcinoma. The average age of the patients was slightly lower in those with small cell carcinoma than in patients with the other types of tumor. The duration of symptoms was shorter in patients with small cell carcinoma than in those with other types of tumor. In all of the cases of small cell carcinoma the patients had symptoms referable to the thorax. Such symptoms were not present in all of the patients with other types of carcinoma. With the exceptions noted, the symptoms were essentially the same in all patients. Roentgenographic examination of the thorax disclosed an abnormality in all but 12 of the total of 1,000 patients. Bronchoscopy was not performed in all patients in the respective groups. It was a very effective diagnostic procedure in those in whom the tumor arose from the central portion of the lung, that is, in cases of small cell carcinoma and in cases of squamous cell carcinoma. Cytological examination of the sputum or bronchial secretions was more effective in patients with small cell carcinoma and least effective in those with adenocarcinoma. The results of surgical treatment were more satisfactory in patients with squamous cell carcinoma than in those with other types of carcinoma. The results of surgical treatment were least satisfactory in patients with small cell carcinoma.

Pulmonary "Coin" Lesions.—Pulmonary "coin" lesions, as defined by Davis and Klepser, are located in the lung substance, and are round, oval or lobulated, with their edges sharply demarcated and contours smooth, without cavitation and usually without calcification. In this type of lesion, a definitive diagnosis usually cannot be confirmed by the various laboratory and x-ray procedures. The diagnosis requires surgical exploration for the removal of the nodule and pathological study. When this is done, these lesions, many of which look similar on the x-ray film, will finally be diagnosed as a large variety of pathological entities. The author presents five illustrative cases in three men and two women between the ages of 34 and 72. Exploratory thoracotomy was performed in all five patients, and the mass was removed. The pathological diagnoses were chronic granuloma (probably due to histoplasmosis), Boeck's sarcoid, tuberculoma, bronchial adenoma, and squamous cell carcinoma of the lung. The important fact is that in various series reported in the literature, from 15 to 30% of the lesions were malignant, and in some specially selected older age groups as high as 70%. Small flecks of calcium, not clearly part of a Ghon complex, do not rule out the possibility that the lesion may be malignant, and they are not a contraindication to exploratory procedures. Exploratory thoracotomy is a benign procedure comparable in risk to appendectomy and should be thought of in the same way as most physicians and even the general public have been educated to think about biopsy of a lump in the breast. The nodule should be removed so that a definite pathological diagnosis can be made. It should not just be watched until a clinical diagnosis can be achieved because by that time metastases are likely to be present and the chance for successful surgery has been greatly reduced. Agreement on these points is almost universal among men skilled in treating diseases of the chest, but is not as widespread among the general medical profession and certainly not among the general public.

Kartagener's Syndrome in Children.—The occurrence of Kartagener's syndrome in four girls and one boy between the ages of 6 weeks and 14 years is reported. Of the five children with transposition of the heart to the right side four had complete situs inversus and one had dextrocardia only. Atelectasis, early or late bronchiectatic changes, and sinusitis were present in all patients. Lobectomy was performed on three with beneficial results. The youngest patient died before the operation could be performed. The author believes that some congenital factor plays a part in the pulmonary features of the disease. It has not been established whether there is a developmental error in the bronchial walls or whether the pulmonary lesions are secondary to the cardiovascular anomalies. It is probable that in most instances atelectasis precedes bronchiectasis. The treatment of Kartagener's syndrome consists of resection of the affected pulmonary tissue as soon as

the patient is deemed a good surgical risk. The age of the patient in the younger years, after the neonatal period, need not be considered in making the decision. Treatment of sinusitis is indicated, although it is usually not nearly so successful as that of the bronchiectasis.

Iowa State Medical Society Journal, Des Moines

43:205-250 (June) 1953

SYMPOSIUM ON BLEEDING FROM UPPER GASTROINTESTINAL TRACT

I: Medical Management. W. M. Kirkendall.—p. 209.
II: Role of Roentgenologist. G. S. Lodwick.—p. 211.
III: Role of Surgery. R. C. Hickey.—p. 213.
IV: Role of Pathologist. K. R. Cross.—p. 215.

Anaphylactic Shock Due to Penicillin. E. A. Larsen.—p. 217.
Allergic Reactions to Catgut Preservatives, Phenol Mercuric Acetate Sensitivity: Case Report. G. S. Rost.—p. 218.
Case of "Pulmonary Adenomatosis" Emphasizing Diagnostic Considerations. L. D. Amick.—p. 219.

Journal Clin. Endocrin. & Metab., Springfield, Ill.

13:629-752 (June) 1953

Effects of Epinephrine on Metabolism of 17-Hydroxycorticosteroids in the Human. A. A. Sandberg, D. H. Nelson, J. G. Palmer and others.—p. 629.
Effect of ACTH and Cortisone on Concentration of Circulating Antibody in Patients with Acute Hepatitis. P. L. Eichman and W. P. Havens Jr.—p. 648.
Comparative Observations Regarding Utilization and Excretion of Infused Glucose, Fructose, and Invert Sugar, Respectively. J. Bertino, N. Dawson, R. French and others.—p. 658.
Effect of Intravenous ACTH on Blood Level of Ascorbic Acid and of Sulfhydryl as Determined by Modified Amperometric Technique. J. W. Goldzieher, P. R. Epstein, W. B. Rawls and M. A. Goldzieher.—p. 662.
Fractionation by Countercurrent Distribution of Urinary Estrogens in Normal Individuals and in Patients with Hyperadrenocorticism. C. J. Migeon.—p. 674.
Comparative Clinical Evaluation of Estrogenic Preparations by Menopausal and Amenorrheal Indices. H. S. Kupperman, M. H. G. Blatt, H. Wiesbader and W. Filler.—p. 688.
Some Metabolic and Biochemical Effects of Methylandrostenediol. F. Homburger, R. M. Dart, C. D. Bonner and others.—p. 704.
Influence of Adrenal and Gonadal Steroids on Uptake of Iodine by Thyroid Gland. W. Zingg and W. F. Perry.—p. 712.
Use of Cortisone for Congenital Adrenal Hyperplasia in the Male: Report of Case. C. F. Gastineau, G. B. Logan, A. Albert and H. L. Mason.—p. 724.

Journal of International College of Surgeons, Chicago

19:665-798 (June) 1953

Compression Granuloma of Spinal Cord Caused by Schistosoma Mansoni Ova; Epiconus; Conus Medullaris; Cauda Equina: Report of Case. C. Gama.—p. 665.
Volkmann's Ischemic Contracture as Complication of Supracondylar Fractures of Humerus. H. W. Meyerding.—p. 675.
Total Pancreatectomy. I. Honjo and C. Araki.—p. 692.
Internal Derangements of Temporomandibular Joint. H. K. Christie.—p. 704.
Diverticulosis and Diverticulitis. S. A. Linde.—p. 716.
*Perirenal Oxygen Insufflation. J. S. Ritter and A. A. Johnson.—p. 732.
Intubated Ureterotomy for Treatment of Stricture of Ureteropelvic Juncture and Upper Part of Ureter: Personal Simplified Technic. C. P. Mathé.—p. 744.
*Surgical Treatment of Mediastinal Lymphoblastoma: Addition of Case and Review of Literature. P. Thorek.—p. 753.
Bone and Joint Changes Following Injury. H. E. Billig Jr.—p. 760.
Effects of Pregnancy on Ulcerative Colitis. O. C. Foster and E. S. Cohen.—p. 763.
Aberrant Pancreatic Tissue in Wall of Stomach. R. G. Majer.—p. 769.

Perirenal Oxygen Insufflation.—Ritter and Johnson point out that the introduction of gases into the perirenal area was first advocated by Carelli. In their own studies they combined Carelli's technique with that more recently described by Rivas. The purpose of the perirenal insufflation is to visualize the structures of the retroperitoneal areas, particularly those lying immediately below the diaphragm. They believe that it is incorrect to say that the Carelli or the direct method of perirenal insufflation is now replaced by the Rivas or the indirect method. These two methods are interrelated; one complements the other. They have found that in cases in which the technique of Rivas failed to reveal the structures of the perirenal area, resort to the Carelli method clearly outlined a tumor of the kidney. Perinephritis and perirenal extravasation due to trauma can be ruled out, when oxygen reaches the renal areas and surrounds the kidney. After experimenting with several gases, the authors found oxygen the most satisfactory and the least dangerous. They describe a new apparatus for accurate measurement of the quantity of gas injected and for recording the pressure under which the gas is introduced. Perirenal insufflation represents a valuable addition to the urologic diagnostic armamentarium.

Surgical Treatment of Mediastinal Lymphoblastoma.—The 10-year-old boy whose history is given had been well until two weeks prior to entrance into the hospital. At that time he complained of pain and "fullness" over the right side of the chest. This was especially marked whenever he attempted to run. Physical examination revealed a slight bulging and diminished breath sounds over the anterior aspect of the right side of the chest. The roentgenogram revealed a mass located in the anterior and middle mediastina. The barium-filled esophagus showed the close proximity of the tumor. The impression was that there was either a dermoid or a teratoma in the anterior mediastinum. The tumor was found to compress the lung, extending from the apex to the right and left innominate veins superiorly, to the pericardium medially and to the superior vena cava posteriorly. Frozen section revealed a lymphoblastoma. It was possible to enucleate the entire mass. The chest was closed and water seal drainage instituted. The postoperative course was uneventful, and a postoperative roentgenogram on the second day revealed good expansion of the right lung. One year has passed since the operation, during which time the boy has been given irradiation therapy. The boy's condition was good at the time of writing this report. In a review of literature 12 similar cases were found in which the patients were operated upon with apparent complete removal of a solitary mediastinal lymphoblastoma. Time and additional experience will be needed to determine whether or not surgical removal of such solitary lesions prior to irradiation will favorably alter the course of this disease.

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Journal of Pediatrics, St. Louis

42:645-794 (June) 1953

Glycogen Storage Disease: Report of Case with Abnormal Glycogen Structure in Liver and Skeletal Muscle. G. B. Forbes.—p. 645.
Glycogen Storage Disease of Liver in Siblings. A. S. Traisman and H. S. Traisman.—p. 654.
*Comparison of Serum Levels Following Administration of Oral and Parenteral Preparations of Penicillin to Infants and Children of Various Age Groups. N. N. Huang and R. H. High.—p. 657.
*Erythromycin in Treatment of Pyoderma in Children. L. S. Freeman and R. B. Scott.—p. 669.
*Use of Banthine Bromide in Infantile Pylorospasm: Report of Nine Cases with X-Ray Study. H. Levy and B. Zweiffler.—p. 673.
Angiography in Case of Hydranencephaly. H. E. Thelander, E. B. Shaw and J. J. Piel.—p. 680.
Calcium Disodium Versenate in Therapy of Lead Encephalopathy. F. E. Karpinski Jr., F. Rieders and L. S. Girsh.—p. 687.
Incidence of Dermatoses Among Infants and Children as Seen in Outpatient Clinic at Skin Hospital in Large City. H. H. Perlman.—p. 700.
*Infectious Hepatitis in Childhood, with Special Consideration of "Progressive Hepatitis." E. S. Murphy and R. B. Johns.—p. 707.
Chest Development in Early Childhood. W. Kornfeld.—p. 715.
New Continuous Nebulizer (Alevizer) for Humidification and Alevaire Therapy. J. B. Miller.—p. 721.
Encephalopathy Occurring During Topical Use of Asterol. N. Goldstein.—p. 726.
Acute Disseminated Lupus Erythematosus with Hemolytic Anemia in 10-Year-Old Child. H. J. Jacobs.—p. 728.
Syndrome of Anomalous Left Coronary Artery. V. C. Kelly, W. S. Wilkins and R. B. Scott.—p. 731.
Substitutes for Cow's Milk in Infant Feeding. J. Glaser.—p. 734.

Serum Levels Following Penicillin Administration.—Penicillin levels in the serum of normal premature infants, normal full-term newborn infants, older infants from 2 weeks to 2 years of age, and children from 2 to 13 years of age were obtained after the administration of the following preparations of penicillin: (1) unbuffered oral potassium penicillin G, administered in a flavored vehicle containing 100,000 units per 5 cc.; (2) oral procaine penicillin administered in a flavored vehicle containing 250,000 units per 5 cc.; (3) crystalline potassium penicillin G diluted with sterile distilled water to

a concentration of 100,000 units per cubic centimeter and administered by intramuscular injection; and (4) procaine penicillin diluted with sterile distilled water to a concentration of 300,000 units per cubic centimeter and administered by intramuscular injection. The various penicillin preparations were given in single doses of 10,000 units per pound of body weight. The penicillin assays were determined by a modification of Rammelkamp's serial dilution method. The test organism was group A hemolytic Streptococcus C-203. Distinctly higher and more sustained blood levels were observed in the full-term newborn and premature infants than in older infants and children after the administration of comparable doses of all four preparations. Lower gastric acidity and inefficient renal function in newborn infants may explain the differences observed between the newborn and older age groups. Minor differences have been observed in the levels following the administration of orally administered potassium penicillin G and orally administered procaine penicillin. Orally administered penicillin in doses of 10,000 units per pound of body weight produced levels of at least 0.03 unit per cubic centimeter for a minimum of six hours in full-term newborn and premature infants, for a minimum of four hours in infants up to 2 years of age, and for at least two hours in children over 2 years old. In all groups, intramuscularly administered potassium penicillin G in doses of 10,000 units per pound of body weight resulted in very high initial levels. The levels declined rapidly, however, so that the six hour levels were comparable to those observed after the oral administration of penicillin in all age groups except the youngest newborn infants. Intramuscularly administered procaine penicillin in doses of 10,000 units per pound of body weight produced high sustained levels for six hours; detectable levels were often demonstrable at the end of 24 hours. The ease with which therapeutically effective levels of penicillin can be achieved in the serum by the oral administration of penicillin indicates definite usefulness of this route of administration in the treatment of infections caused by penicillin-susceptible organisms in newborn and premature infants. A dose of 10,000 units per pound of body weight administered every 12 hours, or 20,000 units per pound of body weight per day, should be effective in the treatment of such infections in full-term newborn and premature infants, while an oral dose of 10,000 units per pound of body weight given at six hour intervals should be adequate for older infants and children. The dosage schedule of parenterally administered penicillin should be guided by the susceptibility of the organism. Beyond early infancy, the doses for infants and children should be comparable as based on body weight.

Erythromycin in Treatment of Pyoderma.—Ten children between the ages of 18 months and 12 years with impetigo and secondary pyoderma were treated with erythromycin ointment (Ilotycin). The ointment was applied locally over the affected areas in all patients. Four patients received erythromycin orally along with the local therapy. In all patients no new lesions appeared after 24 hours of treatment, and crust formation and healing were usually evident in 48 to 60 hours. The average patient received treatment for five days. The healing process continued after the treatment was discontinued. No recurrence developed. Transient hyperpigmentation occurred after healing in most patients. There was no evidence of drug sensitization or other untoward reactions. The courses of the four patients who received oral as well as local treatment with erythromycin were not pronouncedly different from those receiving local therapy alone. The authors feel that erythromycin ointment will prove of value in treating superficial skin infections caused by gram-positive bacteria.

Methantheline in Infantile Pylorospasm.—Eight infants with pylorospasm and one infant with cardiospasm, diagnosed clinically and confirmed by x-ray, were treated with methantheline (Banthine) bromide. The infants with pylorospasm were given 5 mg. of the drug, four times a day, while the one infant with cardiospasm required 50 mg. daily. The usual course of treatment was of one month's duration. All patients that were studied with post-treatment gastrointestinal x-ray series showed demonstrable roentgenographic improvement. Clinically, all eight infants with pylorospasm responded dramatically to methantheline treatment with cessation of vomiting. The infant with cardiospasm and a history of almost daily episodes of forceful vomiting immediately after feeding received methantheline for over two months and had a good clinical result. No toxic effects from the use of the drug were observed. Methantheline bromide is a useful adjuvant to the therapy of infantile pylorospasm.

Infectious Hepatitis in Childhood.—The occurrence of infectious hepatitis is reported in 40 children up to 15 years of age in Denver between 1946 and 1951. There were two deaths of the fulminant type after a total course of nine and eight days respectively in a 5-year-old boy and a 2-year-old girl. Necropsy was performed in both cases, and the liver showed the typical microscopic picture described by Lucké and Mallory. A third death occurred in a 10-year-old boy two months after the appearance of the first symptoms of infectious hepatitis. This patient had a syndrome that was unrelenting from the initial attack and included signs of portal hypertension with ascites, edema, jaundice, and fever. He died a few days after a surgical attempt to ligate his hepatic artery when the ascites had become uncontrollable. The postmortem examination of the liver revealed "subacute necrosis and regeneration," characterized by distortion of the lobular architecture by fibrosis, nodular regeneration of liver cells, and leukocytic infiltration. The 37 remaining patients were considered to have recovered completely from the acute disease. The usual illness began with fever and signs of upper respiratory infection, followed in one to two days by gastrointestinal complaints, including anorexia, nausea, and vomiting. Approximately 12 hours later abdominal pain began. On the fourth or fifth day dark urine or light-colored stools were noted. On the fifth or sixth day jaundice became apparent. Temperature usually was restored to normal with the onset of jaundice. Coupled with this typical clinical story, the laboratory findings of bile in the urine in a high percentage of the patients, an elevated serum bilirubin in all of the patients, an erythrocyte sedimentation rate that was elevated almost without exception, and a normal white blood cell count with a normal differential count confirmed the diagnosis of acute infectious hepatitis in childhood with few exceptions. Jaundice was observed in all of the authors' patients except one, who had the fulminant type and died on the eighth day. The authors believe that there are three clearly defined courses that a case of acute infectious hepatitis may pursue. The first and commonest is the "benign course"; the second, and most disastrous, is "fulminant hepatitis"; and the third, for which the term "progressive hepatitis" is proposed, is the syndrome already described by Alsted, Jersild, Wyllie and Edmunds, that may either end fatally in two to nine months without remission from the initial attack of hepatitis, or may result in at least an apparent clinical cure after this "progressive" period. The authors were unable to find in the American literature reports of cases similar to that of progressive hepatitis with fatal termination in their 10-year-old patient. They agree with Wyllie and Edmunds that the development of signs and symptoms of portal hypertension following an attack of infectious hepatitis in weeks or months is of grave prognostic significance.

Journal Pharmacology & Exper. Therap., Baltimore

108:129-260 (June) 1953. **Partial Index**

Reactions of Strips of Rabbit Aorta to Epinephrine, Isopropylarterenol, Sodium Nitrite and Other Drugs. R. F. Furchgott and S. Bhadrakom. —p. 129.

Comparison of Adrenergic Blocking Action of Ilidar (Ro 2-3248), Regitine (C-7337) and Priscoline in Innervated Saphenous Arterial Bed (Skin Exclusive of Muscle) and Femoral Arterial Bed (Muscle Exclusive of Skin) of Anesthetized Dog. H. D. Johnson, H. D. Green and J. T. Lanier.—p. 144.

Analgesic Power and Question of "Acute Tolerance" to Narcotics in Man. H. K. Beecher.—p. 158.

Colchicine: II. Metabolism of Frog Skeletal Muscle. F. C. Ferguson Jr. —p. 186.

New Group of Potent Sedatives. H. Weidmann and P. v. Petersen. —p. 201.

Cardiovascular Responses During Amphetamine Tachyphylaxis. A. Horita, T. C. West and J. M. Dille.—p. 224.

Journal of Thoracic Surgery, St. Louis

25:545-654 (June) 1953

Contributions of Surgery to Knowledge of Pulmonary Disease. R. M. Janes.—p. 545.
Arterial Oxygen Tension in Rest and During Effort as Test of Pulmonary Function. V. O. Björk, P. A. Michas and L. G. Uggla.—p. 558.
Endobronchial Lesions in Pulmonary Blastomycosis. W. Weisel and F. B. Landis.—p. 570.
Block Dissection in Pneumonectomy for Carcinoma. J. F. Higginson. —p. 582.
Primary Subpleural Intrapulmonic Thymoma. A. R. Crane and P. T. Carrigan.—p. 600.
*Benign Tumors and Cysts of Esophagus. R. C. Totten, A. P. Stout, G. H. Humphreys and R. L. Moore.—p. 606.
*Surgical Correction of Atrial Septal Defects Associated with Transposition of Pulmonary Veins. W. B. Neptune, C. P. Bailey and H. Goldberg. —p. 623.

Benign Tumors and Cysts of Esophagus.—Most of the data reviewed in this paper were collected from the reports of 163 cases of tumors and cysts. Some were clinically manifest, and others were incidental autopsy findings. Forty other cases were reviewed and discarded because of incomplete clinical or pathological information. The clinical material includes 40 intraluminal polypoid tumors, 46 solid intramural tumors, and 17 cysts. The sessile intraluminal growths, supernumerary tissues, muscular hypertrophy, and inflammatory conditions are discussed in the section on pathology, since many did not produce symptoms. Benign tumors of the esophagus may be divided into two groups: the intraluminal and the intramural. In the former group most of the tumors are fibrous polyps, although polypoid lipomas and leiomyomas occur. The majority of these intraluminal tumors are attached in the region of the cricoid cartilage. Sessile intraluminal growths occur, but rarely, if ever, are of clinical importance. The solid intramural tumors are predominantly leiomyomas and may be found at any level. The intramural cysts are congenital in origin and usually contain derivatives of the primitive foregut. Most of these occur in the middle or lower thirds of the esophagus. Supernumerary tissues, other than gastric epithelium, rarely appear in the esophagus and inflammatory conditions are not common. The tumors, particularly the polyps, may be difficult to diagnose, but roentgenography and esophagoscopy are helpful. Most of the polypoid tumors may be removed via the esophageal lumen, whereas a thoracic or abdominal approach plus enucleation and/or resection is necessary to treat the intramural growths. Malignant polypoid and intramural tumors occur that clinically can mimic the benign growths. A good prognosis may be expected provided an accurate diagnosis is made and proper therapy is employed.

Surgical Correction of Atrial Septal Defects.—Among their patients with atrial septal defects, Neptune and associates observed five with anomalous drainage of the veins of the right lung into the right atrium, just to the right of the remnant of the atrial septum. None of these defects were of the persistent ostium primum type; all were located posteriorly in the septum, although they varied in their distance from the auriculoventricular valves. They were relatively large, even in the children, and capable of admitting one to three fingers. Among the various methods that have been tried or suggested for the closure of atrial septal defects, Bailey's atrioseptopexy technique was the first effecting complete surgical closure of large defects of this type. Neptune and his associates used successfully a simple modification of this atrioseptopexy technique in two of five cases that were observed since this technique was developed. Detailed case reports of these two patients with septal defects and transposition of the pulmonary veins of the right lung are described. Successful repair of the defects was proved by cardiac catheterization. The technique for the complete surgical correction of atrial septal defect associated with transposition of the pulmonary veins of an entire lung is presented. A method for correction of anomalous pulmonary venous drainage of all pulmonary veins into the right atrium and/or its tributaries is suggested.

Medical Annals of District of Columbia, Washington

22:279-338 (June) 1953

Role of Some Experimental Anticonvulsants, Mysoline, Milontin and 1461L. B. Smith and F. M. Forster.—p. 279.
Some Notes on Antibiotics and Chemotherapy in Infectious Diseases. W. J. Martin.—p. 283.
Selection of Patients for Psychiatric Referral. M. L. Adland.—p. 289.
Thyroiditis. J. H. Lyons.—p. 292.
Endometriosis. R. Bieren.—p. 298.

Michigan State Medical Society Journal, Lansing

52:569-688 (June) 1953

Jaundice. P. Thorek.—p. 597.
Recurrent Acute Intussusception. J. E. Summers.—p. 601.
Psychodynamic Aspects of Peptic Ulcer. L. A. Schwartz.—p. 603.
Cortisone and Pregnancy. E. J. De Costa.—p. 613.
Epilepsies: Their Diagnosis and Treatment. R. P. Mackay.—p. 618.
Health Study of Detroit-Windsor Air Pollution Investigation. J. G. Molner and A. S. Rabson.—p. 626.
The Acutely Inflamed Eye. P. C. Kronfeld.—p. 631.
Diagnosis of Occupational Disease. O. T. Mallery Jr.—p. 633.

Missouri Medicine, St. Louis

50:395-462 (June) 1953

*Amniotic Fluid Pulmonary Embolism: Report of Two Cases and Review of Literature. J. C. Bacala.—p. 411.
An Introduction for Physicians to an In-Patient Rehabilitation Service in General Hospital. O. Machek.—p. 415.
Selection of Far Advanced Cancer Patient for Roentgentherapy. J. O'Donoghue.—p. 419.
*Weber-Christian Disease: Report of Two Cases Treated with Cortisone. L. B. Harrison and J. A. Saxton Jr.—p. 427.
Misconceptions Concerning Nuclear Explosions. C. P. Hungate.—p. 431.
Good Physicians, Good Citizens. G. S. Benson.—p. 435

Amniotic Fluid Pulmonary Embolism.—Pulmonary embolism caused by amniotic fluid is a rare entity, only 44 cases having been reported since it was first described by Steiner and Lushbaugh in 1941. The condition is characterized clinically by sudden shock during labor in a previously asymptomatic woman. Its cause may be strong or tetanoid contractions, administration of pitocin, quinine or castor oil, an oversize fetus, prolonged intervals between deliveries, multiparity, previous cervical laceration and conization, and sometimes obstetric abnormalities. Diagnosis is based on the clinical picture; autopsy may reveal a ruptured uterus, pulmonary edema, edema and hemorrhages in the broad ligament, retroperitoneal hematoma and hemorrhage, and embolism. Histological examination may show amniotic fluid elements, such as epithelial squamae, meconium, vernix caseosa, mucinous debris, and lanugo in the pulmonary vessels. Although death has been ascribed to reflex vasospasm or mechanical occlusion and anoxia, anaphylaxis seems to be the most likely cause. Late studies have revealed that normally the amniotic fluid passes through the placental barrier, enters the maternal circulation, and is excreted through the kidneys. Thus, primary sensitization of the mother to the amniotic fluid is explained, and, when the amniotic fluid enters suddenly into her blood stream during the shock, anaphylaxis ensues. The authors report two cases of amniotic fluid pulmonary embolism in a total of 26,322 deliveries at the obstetrical department of St. Anthony's Hospital in St. Louis during the last 10 years. The diagnosis was established in both patients on the basis of the clinical picture. In the first patient, a 35-year-old woman, para 5, with a previous cervical conization, a ruptured uterus and amniotic fluid extravasation were found at autopsy; histological examination of the lungs revealed amniotic fluid particulates in the pulmonary vessels. In the second patient, a 41-year-old woman, para 7, microscopic examination did not show amniotic fluid constituents in the lungs but autopsy revealed emboli in the bifurcation of the pulmonary artery and extravasation of amniotic fluid in the left pelvic floor adjacent to an area of uterine softening that possibly may have been a uterine tear.

Cortisone in Nodular Nonsuppurative Panniculitis.—Results are reported of cortisone therapy in two women with nodular nonsuppurative panniculitis. The disease in both patients was

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confined to the lower legs, although as a rule the subcutaneous tissues of other parts of the body are also involved. Both legs were greatly swollen in the first patient, and tender nodular swellings were present beneath the skin in the calves. Despite administration of antibiotics and sulfadiazine, the number of nodules continued to increase and the pain increased to such an extent that the patient was confined to a wheel chair. Cortisone therapy was then attempted; a dose of 50 mg. was given at first four times daily, then, after the first improvement, three times daily, and finally twice daily. Within two weeks the swelling had disappeared from both legs, and only one or two small but not tender nodules were still present in each calf. The patient was discharged and continued to take the drug for four weeks. After four months she was still well and was doing her own housework. A relapse occurred six months after she left the hospital, but the recurrent symptoms subsided again with cortisone therapy. Administration of 50 mg. of the drug four times daily for 10 days brought about a prompt improvement in the other patient. Four months later she was well and only small depressions over the posterior surface of each calf could be seen. Cortisone is valuable in the treatment of nodular nonsuppurative panniculitis but it is not a cure. The cause of this disease is still unknown. Observations in these two cases suggest that it probably is not a local bacterial infection and that the tissue reactions that cause the onset of the symptoms are suppressed by cortisone in the same manner that this drug suppresses the tissue reactions in some collagen diseases. A relationship to collagen diseases is suggested by the similar response to cortisone therapy and the relapsing course of nodular nonsuppurative panniculitis, but further evaluation of cortisone therapy in this disease is needed.

New England Journal of Medicine, Boston

248:959-994 (June 4) 1953

SYMPOSIUM ON OBESITY

Reorientation on Obesity. A. W. Pennington.—p. 959.
Lipogenesis. S. Gurin.—p. 965.
Health and Obesity. D. P. Barr.—p. 967.
Relation of Obesity to Longevity. L. I. Dublin.—p. 971.
Psychology of Overeating. H. W. Brosin.—p. 974.
Treatment of Peptic Ulcer. L. Betzel.—p. 976.

Oral Surgery, Oral Medicine & Oral Path., St. Louis

6:691-804 (June) 1953. **Partial Index**

Open Reduction of Fractures of Mandible. R. E. Crowley.—p. 691.
Incidence of Gingivitis Among Sample of Massachusetts School Children. D. G. Stahl and H. M. Goldman.—p. 707.
Studies in Etiology of Chronic Desquamative and Hyperkeratotic Lesions of Oral Mucous Membranes. M. Karshan, D. E. Ziskin, H. F. Silvers, G. Stein and A. H. Kutscher.—p. 716.
Clinical Toxicity of Epinephrine Anesthesia. S. P. Dick.—p. 724.
*Multiple Myeloma Occurring in Jaws: Study of 17 Cases. K. W. Bruce and R. Q. Royer.—p. 729.
Hyperparathyroidism (Generalized Osteitis Fibrosa). W. Schneider. —p. 745.
Cysts of the Gingiva. B. Ritchey and B. Orban.—p. 765.
*Osteogenesis Imperfecta Tarda with Dentinogenesis Imperfecta. P. D. Toto.—p. 772.

Multiple Myelomas Occurring in the Jaws.—The case records and roentgenograms of the jaws of 59 patients with myeloma were reviewed. Of these patients, 17 exhibited evidence of myeloma in the jaws. Whereas the ratio of males to females was approximately 2 to 1 in the 59 patients, among the patients with lesions in the jaws women predominated. All but one patient with myeloma of the jaws had general skeletal involvement; this patient had a solitary myeloma of the jaw and no lesions elsewhere in the skeleton. The mandible was more frequently involved than the maxilla. Lesions occurred oftener in the posterior portion of the body, the angle, and the ascending ramus of the mandible than elsewhere in the jaw. More patients exhibited lesions in the skull than in the jaws. Symptoms referable to the jaw, such as pain, pressure sensation, numbness, swelling, expansion of the jaw, mobility of teeth, and epulis formation, were observed. In seven cases the initial symptoms of myeloma were observed first in the jaw. Usually myelomas of the jaw appear roentgenographically as multiple rounded lesions that have little circumferential osteosclerotic bone reaction. These vacuolated configurations are frequently situated within a larger region of diffuse radiolucency with hazy ill-defined margins. Histopathologic examination of tissue from the jaw in 5 cases disclosed tumor tissue that was composed of closely packed anaplastic cells resembling plasma cells. The supporting stroma was scanty but vascularity was prominent. This study demonstrates the prevalence of myelomas in the jaw and illustrates that oral examination can be helpful in the diagnosis of generalized disease.

Osteogenesis Imperfecta with Dentinogenesis Imperfecta.—Osteogenesis imperfecta, osteopsathyrosis, fragilitas ossium, blue sclera and brittle bones are synonymous. Dentinogenesis imperfecta, odontogenesis imperfecta, and hereditary opalescent dentine are synonyms of allied dental disturbance, which is characterized by chipping and cracking of the enamel with a rapid wearing down of the teeth, and an opalescent color described as blue, gray, and reddish-brown. The case presented is one of osteogenesis imperfecta tarda illustrating the typical physiognomy, history, and allied dental disturbance. The patient was a 17-year-old youth, who complained that the enamel of his teeth was chipping off. The teeth were opalescent gray-brown. The physiognomy was characterized by bitemporal protuberances with the ears turned out and forward. The forehead appeared broad, but the maxilla and mandible appeared small. This gave a triangular shape to the face. When questioned regarding the history of fractures, the patient admitted having suffered fractures of the left clavicle five times, of the left humerus once, and of the little finger on the left hand once.

Psychiatric Quarterly, Utica, N. Y.

27:185-366 (April) 1953

Concerning Nature of Communication. E. Berne.—p. 185.
Three-Year Follow-Up Study of Nonconvulsive Histamine Biochemotherapy, Electric Convulsive Post-Histamine Therapy, and Electric Convulsive Therapy Controls. R. R. Sackler, A. M. Sackler and M. D. Sackler.—p. 199.
Factors Involved in Genesis and Resolution of Neurotic Detachment. M. Ullman.—p. 228.
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Treatment Plan Combining Group and Individual Psychotherapeutic Procedures in State Mental Hospital. J. Green.—p. 245.
Histamine Therapy in Psychiatric Disorders. P. Polatin, A. S. Effron and R. C. Robertiello.—p. 254.
Dream Life in Case of Hebephrenia. B. Karpman.—p. 262.
Ligation of Anterior Choroidal Artery for Involuntary Movements—Parkinsonism. I. S. Cooper.—p. 317.

Psychiatry, Washington, D. C.

16:99-214 (May) 1953. **Partial Index**

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Quart. Bull. Sea View Hospital, Staten Island, N. Y.

14:49-96 (April) 1953

Treatment of Advanced Caseous-Pneumonic Tuberculosis with Isonicotinic Acid Hydrazides. I. G. Tchertkoff, M. Asisian and P. Nenashev. —p. 49.
Results Obtained with Streptomycin and Other Modalities in 154 Patients with Pulmonary Tuberculosis. G. G. Ornstein and L. Lercher.—p. 57.
*Isonicotinic Acid Hydrazide in Excisional Surgery of Tuberculous Lung. I. R. Davidson and M. Beaulieu.—p. 66.
Treatment of Pulmonary Hydatid Cysts: Experience with Over 500 Cases in Greece. E. Lecos.—p. 72.
Case of Chronic Pulmonary Tuberculosis Treated with Iproniazid and Streptomycin: Pathology Report. V. Altmann and E. Shin.—p. 78.
Chronic Pulmonary Tuberculosis Complicated by Hodgkin's Disease and Treated with Isoniazid and Streptomycin: Autopsy Findings in a Case. V. Altmann, A. Tendlau and E. Shin.—p. 87.

Isoniazid in Pulmonary Excision for Tuberculosis.—Preliminary observations are presented on the use of isoniazid and iproniazid in excisional surgery of the tuberculous lung and

in the treatment of complications of resection. Twelve patients received the isoniazid preoperatively, and a second group of 28 patients received it only for postoperative complications. The authors gained the impression that, with the use of the isonicotinic acid derivatives, lung resection may be made available to more patients with far advanced pulmonary tuberculosis than was hitherto considered possible. Operation may be done for lesions that would ordinarily be considered inoperable. Complications that follow resection may be managed better with the aid of these drugs.

Review of Gastroenterology, New York

20:353-448 (June) 1953

SYMPOSIUM FOR THE GENERAL PRACTITIONER

Acute Lower Abdominal Emergencies. D. B. Crawford Jr., W. A. Wickern and H. W. Cave.—p. 363.
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Milestones in Diagnosis and Treatment of Diarrheal Diseases. W. Z. Fradkin.—p. 410.
Problems in Differential Diagnosis of Jaundice. C. M. Leevy and J. A. Emmert.—p. 422.

Rhode Island Medical Journal, Providence

36:229-284 (May) 1953

Medicolegal and Pathological Aspects of Suicide. A. E. O'Dea.—p. 245.
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Videotherapy as an Office Procedure. B. B. Mongillo.—p. 253.

Rocky Mountain Medical Journal, Denver

50:447-530 (June) 1953

Surgery of Major and Minor Traumatic Wounds Including Burns. H. Conway.—p. 469.
X-Ray Diagnosis of Neoplasms of Gastrointestinal Tract. R. J. Bloor. —p. 475.
Clinical and Diagnostic Aspects of Carcinoma of Lung. M. E. Murphy. —p. 480.
Aldrin Poisoning. E. Nelson.—p. 483.

Southern Medical Journal, Birmingham, Ala.

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Concept and Clinical Aspects of Factitial Dermatitis. D. T. Gandy. —p. 551.
*Advantages of X-Ray Diagnosis in Pregnancy. R. H. Barter and J. Parks. —p. 555.
Report on Cytology of 5,800 Vaginal Smears. L. F. Turlington, W. C. Barclift and H. M. Brown.—p. 562.
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Continuous Enteral Feeding and Its Importance in Protein Metabolism of the Sick. J. Elliott, J. J. Griffitts, D. W. Smith and others.—p. 572.
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Differentiation and Management of Abnormal Mental States in the Aged. W. T. Brown and J. A. Smith.—p. 582.
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Treatment of Tendon Injuries. S. B. Fowler, D. L. Eyler and G. K. Carpenter.—p. 601.
Polyradiculopathy in Man Produced by St. Louis Encephalitis Virus (SLE). M. Sanders, A. Blumberg and W. Haymaker.—p. 606.
Recurrent Henoch-Schonlein Purpura. A. M. Freeman.—p. 609.
Cortisone in Ophthalmology: Clinical Evaluation of Therapeutic Results. L. A. Breffeilh.—p. 612.
Electro-Encephalography in Allergic Epilepsy. S. C. Dees.—p. 618.
What the General Practitioner Should Know About Management of Allergic Skin Disorders. W. M. Sams.—p. 620.

Advantages of X-Ray Diagnosis in Pregnancy.—Barter and Parks believe that even though x-ray diagnosis is being used oftener in obstetrics, relatively few obstetricians utilize its full potentialities. Discussing roentgenologic pelvimetry, the authors say that, while ideally all primigravidas should be subjected to x-ray pelvimetry, as a rule it is done only for those in whom abnormalities are revealed by manual mensuration. Such abnormalities include a diagonal conjugate of 11.5 cm. or less, a reduction in the interspinous and intertuberous diameters, abnormal curvature or forward displacement of the sacrum, and the characteristics of the masculine type of pelvis. Roentgen ray pelvimetry is indicated also in primigravidas with a breech or other abnormal presentation or with an unengaged fetal head at term. Roentgen ray pelvimetry is indicated in all pregnant women who are less than 5 ft. (152 cm.) tall, who have an abnormal presentation, particularly if the fetus seems larger than previous ones, who have a history of difficult labor or of previous cesarean operation, whose labors are progressing poorly, or who have a history of a pelvic fracture. The presence of true uterine inertia should never be accepted unless cephalopelvic disproportion has been ruled out as a cause of ineffective labor. Aside from pelvimetry, roentgen examination is helpful in determining the presentation and position of the fetus and in the diagnosis of multiple pregnancy. In the presence of an overdistended uterus, roentgen examination is important to differentiate between polyhydramnios, a single large fetus, or a multiple gestation. In the late weeks of pregnancy the age of the fetus may be determined to some degree. Antepartum diagnosis of an anomalous fetus can be made in most instances. Fetal death in utero may be established by roentgen rays, although the findings may not become apparent until several days after the death of the fetus. Placentography has been used successfully in some clinics, and the diagnosis of abdominal pregnancy can be facilitated by the use of roentgen rays. The roentgenologist and obstetrician must work as a team if the best results are to be obtained with roentgen examination in the care of the maternity patient and her child.

Southwestern Medicine, El Paso, Texas

34:149-188 (May) 1953

Current Medical Problems for 1953-1954. G. Turner.—p. 162.
Notes from Orthopaedic Surgery: Fractures: Non Unions. W. C. Basom. —p. 171.

34:189-228 (June) 1953

Care and Treatment of Congestive Heart Failure. R. H. Homan.—p. 208.
Differences Between Collagen Diseases. L. G. Jekel.—p. 212.

Stanford Medical Bulletin, San Francisco

11:79-136 (May) 1953. **Partial Index**

*Nontropical Sprue Treated Successfully with Corticotropin: Case Report. B. M. Meyer and A. Goldfien.—p. 91.
Microdiffusion Method for Alcohol Determination. E. J. Newman and H. W. Newman.—p. 96.
Precipitating Stresses in Peptic Ulcer. T. M. Myers.—p. 100.
Recent Advances in Radiation Therapy. L. H. Garland.—p. 110.
Benign Tumors of Parotid. R. S. Pollack and D. K. Clawson.—p. 115.
Atypical Blood Clotting Patterns in Hemophilia. T. H. Spaet, B. Kinsell, H. M. Behring and P. M. Aggeler.—p. 118.

Corticotropin in Treatment of Nontropical Sprue.—The diagnosis of idiopathic steatorrhea in the patient described was supported by the presence of diarrhea, steatorrhea, a flat oral glucose tolerance test curve, megaloblastic anemia, and free hydrochloric acid in the gastric juice. The patient had previously responded to treatment with crude liver extract, folic acid, and vitamin B_{12}, but the cessation of therapy was followed by a relapse. At the time of writing the patient has been in clinical and hematological remission for more than 15 months following treatment with corticotropin. The corticotropin was given by intravenous infusion for 54 days. For the first 18 days of treatment he received 13.5 units of corticotropin daily. The dosage was then reduced to 10 units daily for 6 days, after which it was reduced to 5 units daily for 30 days. Adequate adrenal response to treatment was proved by the fall in fasting eosinophil counts. Throughout the period of treatment the patient was maintained on a high calorie, low salt diet with supplemental potassium. The response of this patient to corticotropin supports previous observations that adrenal steroids alter the course of idiopathic steatorrhea.

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The more pronounced and prolonged effect of therapy in this case, as compared to those previously reported, may be in part due to more vigorous adrenal stimulation. Although observations published to date indicate that endogenous or exogenous adrenal steroids can alter the pathological physiology of idiopathic steatorrhea, the nature of this alteration and the manner in which it takes place is unknown. It is pointed out that adrenal insufficiency and steatorrhea may have the following disturbances in common: asthenia, hyperpigmentation, hypotension, steatorrhea, and an abnormal blood sugar response to ingested glucose. Recent studies fail to support the possibility that a causal relationship exists between adrenal insufficiency and sprue. It is assumed that the response to corticotropin in this patient was due to improved intestinal absorption of various nutrients including hematopoietic factors.

Surgery, St. Louis

33:799-966 (June) 1953

Prediction of Effect of Lumbar Sympathectomy on Skin Temperature of Lower Extremity in Patients with Obliterative Arterial Disease. J. J. Cranley, S. A. Leiseca, J. L. Buchanan and others.—p. 799.
*Role of Sympathectomy in Treatment of Frostbite. N. H. Isaacson and J. B. Harrell.—p. 810.
Experiences with Epidural Analgesia for Sympathetic Block. J. R. Thistlethwaite, T. G. Edison, C. Gruenwald and I. Harrison.—p. 818.
Some Effects of Cortisone on Aortic Grafts. J. R. Barberio, J. W. Pate, P. N. Sawyer and C. A. Hufnagel.—p. 827.
Method for Complete Drainage of Superior and Inferior Venae Cavae into Extracorporeal Circuits. J. A. Helmsworth, L. C. Clark Jr., R. T Sherman and others.—p. 835.
Study of Influence of Adrenal Steroids on Strength of Healing Wounds: Preliminary Report. E. C. Meadows and J. F. Prudden.—p. 841.
Recanalization of Vena Cava: An Experimental Study. R. M. Miles and J. M. Young.—p. 849.
Surgical Treatment of Mitral Insufficiency: Experimental Use of Transplanted Pericardium in Dogs. A. R. Henderson and C. L. Law.—p. 858.
Nature of Bleeding in Esophageal Varices. T. W. Wagenknecht, J. F. Noble and I. D. Baronofsky.—p. 869.
*Hypothalamic Influences on Hydrochloric Acid Secretion of the Stomach. R. W. Porter, H. J. Movius and J. D. French.—p. 875.
Spontaneous Fistula of Abdominal Wall with Chronic Pancreatitis, Abscess Formation, and Calculi. A. M. Vaughn and W. B. Hobbins. —p. 881.
Radical Groin Surgery with Particular Reference to Postoperative Healing. J. P. Woodhall.—p. 886.
Prevention of Pancreatic Fat Necrosis by Enzyme Inhibitors: III. Quinine. H. L. Popper and H. Necheles.—p. 896.
"Rectus-Wick" Operation in Ascites. G. G. Crowe.—p. 898.

Sympathectomy in Treatment of Frostbite.—A unilateral ganglionectomy was performed in 28 men and 5 women between the ages of 22 and 65 who had bilaterally equal frostbite in both lower extremities. The untreated extremity served as a control for evaluation of the sympathectomy. Most of the patients sustained their injury after an alcoholic bout on a cold, wintry night. In 24 patients ganglionectomy was performed prior to the onset of gangrene, while the remaining 9 patients already had gangrene of varying extent involving their lower extremities. Sympathectomy did not prevent or limit the extent of gangrene when performed early, nor did it hasten healing once gangrene was established. Seven patients of the 33 had 10 subsequent admissions to the hospital for repeated frostbite, which was usually sustained in a manner similar to their initial injury. Sympathectomy did not protect the extremity against repeated cold injury. Seven patients presented themselves 3 months to 31 years after cold injury with one or more of the chronic sequelae such as paresthesias and pain, trophic ulcers, and coldness and numbness of feet. It is in the treatment of these late sequelae of frostbite that sympathectomy has its greatest application. In all patients the feet became subjectively and objectively warmer following ganglionectomy, the skin temperatures increasing 2 to 4 degrees Fahrenheit by actual measurement and oscillometric readings by up to 40%. Paresthesias and pain were relieved. Ulcers disappeared, circulation improved, and symptoms subsided. As proof of the efficacy of the procedure, results were so impressive that sympathectomy was proceeded with in the control extremity in five patients, with similar spectacular results.

The Hypothalamus and Hydrochloric Acid Secretion.—Experiments were made on monkeys to study alterations in gastric hydrochloric acid secretion induced by stimulation of the hypothalamus. The animals were anesthetized with cyclopropane and concentric bipolar electrodes were placed in the anterior or posterior hypothalamus after small burrholes were made in the cranium. Condenser discharge stimuli of 2 v. were delivered at a frequency of five per second for 30 seconds. Gastric samples were obtained through a Levin tube before stimulation and at half hour intervals for five hours thereafter, and their hydrogen ion concentrations were determined with a glass electrode pH meter. Animals had been fasted for 24 hours and the stomach emptied as completely as possible before stimulation. The anterior or posterior hypothalamus was stimulated in intact animals, 48 hours after bilateral vagotomy, 48 hours after bilateral adrenalectomy, 48 hours after bilateral adrenalectomy and vagotomy, and 48 hours after spinal cord section. In a second series of experiments, the gastric hydrochloric acid response to subcutaneous insulin and corticotropin (ACTH) was measured in intact animals 48 hours after bilateral vagotomy, and 48 hours after bilateral adrenalectomy. Results of the stimulation experiments revealed that the hypothalamus increases the secretion of gastric hydrochloric acid by two distinct and separate routes. The first originates in the anterior hypothalamus, is conducted through the vagus nerve, and is blocked only by vagotomy. The peak of its response is prompt. The second, with a later peak response, arises in the posterior hypothalamus, is conducted humorally through the pituitary-adrenal system, and is blocked only by adrenalectomy. The increase in gastric hydrochloric acid induced by administration of insulin, exhibits features of the responses to both anterior and posterior hypothalamic stimulation. It would appear, therefore, that insulin excites hydrochloric acid secretion through both channels. The administration of corticotropin to animals not operated on produced an increase in gastric acid secretion that was very similar to that resulting from posterior hypothalamic stimulation. Vagotomy had no effect on this response, while adrenalectomy abolished it completely. These data on the monkey may be expected to approximate more closely those concerning human physiological processes than others previously reported in carnivores. The authors' observations support those described in the cat and dog, and hence strengthen extrapolations of these experimental findings in man.

Tennessee State Medical Assn. Journal, Nashville

46:197-234 (June) 1953

Ideals and Ethics of Medicine. P. R. Hawley.—p. 197.
Management of Headache. E. P. Cutter.—p. 202.
Diagnosis and Treatment of Patent Ductus Arteriosus. W. K. Swann, T. L. Lomasney and J. E. Acker.—p. 205.
Gynecomastia. J. G. Riddler.—p. 210.
Case Report: Mixed Tumor of Hypopharynx with Malignant Changes and Distant Metastasis. H. Duncan.—p. 212.

Texas State Journal of Medicine, Fort Worth

49:351-484 (June) 1953

Looking Back. T. C. Terrell.—p. 354.
Current Medical Problems for 1953-1954. G. Turner.—p. 356.
Crossing the Bar. L. H. Reeves.—p. 359.
What is Public Relations? G. Turner.—p. 360.
What Your County, State, and National Medical Organization Can Do for You. E. J. McCormick.—p. 362.
Public Relations Responsibilities of County Society. R. W. Waterson. —p. 364.
The Physician, His Wife, and Government. R. B. Chrisman Jr.—p. 366.
The Patient and Proper Function of the Doctor's Office. E. Hess, R. B. Roth and A. F. Kaminsky.—p. 368.
Advising Patients Regarding Travel in Latin America. J. Baker. —p. 371.
*Pleurodynia Outbreak in North Texas. J. A. Risser.—p. 374.

Pleurodynia Outbreak in North Texas.—Risser first recognized pleurodynia in Fannin County of northeast Texas in August, 1951, when a 35-year-old druggist telephoned at 4 a. m. saying that he could not breathe and that he was having a heart attack. Examination revealed a zone of pain with numbness along the right costal margin. Fluoroscopy showed an elevated and splinted diaphragm with no pneumothorax. The heart and pleurae were normal. Intramuscular injection of meperidine (Demerol) gave considerable relief. Another

paroxysm of pain along the left costal margin was again accompanied by respiratory distress. A son, aged 3, and a daughter, aged 5, had experienced mild febrile illness with paroxysmal respiratory distress only a few days before. Looking back over records of patients seen in July, Risser found several faulty impressions: herpes zoster without eruption, coronary occlusion without electrocardiographic signs, gallbladder disease without jaundice or stones, and duodenal ulcer without roentgen ray confirmation. In a number of other patients who complained that "it hurts to breathe," he had arrived at no diagnosis, but by Aug. 7, a number of patients with this complaint were observed daily and other local physicians had dealt with similar cases. With the aid of the state health department and of a team of the National Institute of Health, studies were made on specimens of feces, blood, and throat washings. The viruses isolated from the initial group of cases, were Coxsackie B, type 3. Since this agent was isolated from the stools of 16 of 17 selected uncomplicated cases of pleurodynia in which there was evidence of antibody response, it is thought that the Coxsackie B, type 3 virus was the cause of this outbreak. The last classical cases of pleurodynia were seen in October. Counting the misdiagnosed cases of July in addition to those properly called pleurodynia, the author personally examined and prescribed for 128 cases of clinical pleurodynia. The author stresses the extreme contagiousness, pointing out that most laboratory workers who handle the virus succumb to pleurodynia. He also mentions that at the conference on poliomyelitis held in Denmark it was suggested that immunity to "devil's grip" (pleurodynia) viruses might protect against poliomyelitis.

U. S. Armed Forces Med. J., Washington, D. C.

4:795-950 (June) 1953. **Partial Index**

Treponemal Immobilization Test in Diagnosis of Syphilis. F. W. Chorpenning.—p. 807.
Tumors of Anterior Cervical Region. H. C. Spear.—p. 821.
Clinical Review of Thirty Cases of Carcinoma of Pancreas. J. C. Country and R. Foulk.—p. 831.
Psychiatric Social Work in Combat Area. R. W. Morgan.—p. 847.
Vasomotor Rhinitis Syndrome. J. A. Budetti.—p. 857.
*Adolescent Kyphosis. T. E. Dameron Jr. and W. H. Gulledge.—p. 871.
Repair of Traumatic Finger Tip Amputations. J. L. Holmes.—p. 877.
Electrocardiography and the General Practitioner. J. E. Goodman. —p. 889.
Essential Hypertension: Treatment with Hexamethonium and Apresoline. R. P. Shapera and F. R. Lang.—p. 903.
Therapeutic Pneumoperitoneum. E. C. Casey and K. W. Clement. —p. 921.
Myoblastoma of Tongue. J. E. Chipps and G. R. Reinhardt.—p. 925.

Adolescent Kyphosis.—In the course of the examination of 1,000 young men at a recruit-training center, Dameron and Gulledge found 83 with a significant dorsal kyphosis which could not be corrected by active or passive attempt. These men were then studied more closely with a complete history, physical examination, and lateral roentgenograms of the thoracic and lumbar vertebras. Roentgenographic criteria for a diagnosis of adolescent kyphosis were: (1) anterior wedging of two or more thoracic vertebrae, (2) increased anteroposterior diameter of the affected vertebrae, and (3) irregularity of the adjacent vertebral surfaces. Twenty-two of the 83 patients did not have vertebral changes sufficient to justify the diagnosis of adolescent kyphosis, and so there remained 61 among 1,000 healthy young men who had the deformity. Adolescent kyphosis has been discussed also under such terms as Scheuermann's disease, juvenile dorsal kyphosis, and vertebral epiphysitis. Reports suggest that it occurs chiefly in males (70 to 90%). The general health of the patient is usually good and the musculature shows normal development. There may be pain in the area of the kyphosis for a few weeks early in the course of the disease. The deformity, noticed by relatives or friends, is the most frequent complaint. The sixth through the 10th thoracic vertebrae are involved most frequently. The treatment generally recommended is support of the spine by casts, braces, or bed rest to prevent accentuation of the kyphos. Results have been inconclusive. No treatment is indicated after vertebral growth has stopped.

Virginia Medical Monthly, Richmond

80:301-354 (June) 1953

Osler and Welch Founders of Modern American Public Health. H. Williams.—p. 303.
*Duodenal Diverticula. G. W. Horsley and G. J. Vlasis.—p. 313.
Resection Reconstruction of Femoral Head. E. D. Vere Nicoll.—p. 319.
Pancreatitis. J. R. Massie Jr.—p. 322.
Galactosemia. McL. Birdsong and J. B. Wood.—p. 327.
Napoleon's Views on Complications of Childbirth. C. D. Bradley.—p. 330.
Carcinoma of Pancreas—Clinical and Laboratory Observations in 29 Proven Cases. J. O. Burke and K. Plummer.—p. 333.

Duodenal Diverticula.—Horsley and Vlasis review observations on 43 patients with duodenal diverticula seen at a Richmond, Va., hospital during the past 10 years. More of the diverticula occurred on the concave side of the descending duodenum, and the majority of these were false. The incidence of diverticula occurring in each portion of the duodenum in these 43 patients was as follows: in the ascending portion, 12; in the descending portion, 20; in the transverse portion, 3; in the terminal portion, 1; between the ascending and descending portions, 3; between the descending and transverse portions, 1; and between the transverse and terminal portions, 3. There were approximately as many men as women. Five of the 12 patients in whom the duodenal diverticulum was the only lesion were treated by surgical excision and improved subsequently. The remaining patients were treated medically, since the severity of their symptoms did not warrant surgical interventions or they were not suitable candidates for surgery. The remaining 31 had an associated duodenal ulcer, cholecystitis, cholelithiasis, diverticula of the colon, and/or other gastrointestinal disorders. In one case of duodenal diverticula and duodenal ulcer, the diverticula were felt to be the major lesion, and the patient was treated by excision of the diverticula and vagotomy, with relief of symptoms. Once the diagnosis has been established by roentgenologic examination, the question arises whether medical or surgical treatment is required. The symptoms observed oftenest are those either referable to the biliary tract or suggestive of duodenal ulcer. The predominant symptom is pain above and to the right of the umbilicus. The patient may also have headache, nausea, distention, water brash, vomiting, and other symptoms suggestive of biliary dyskinesia. This pattern of symptoms referable to the biliary tract probably results from the fact that usually the diverticulum is in the same segment as the biliary tract. Pressure over the diverticulum may cause radiation of pain posteriorly to the back and/or below the costovertebral angle. The authors feel that surgical treatment is indicated under the following conditions: when the symptoms persist despite conservative therapy; when barium is retained in the diverticulum for four hours or longer; when the diameter is more than 3 cm.; and when the diverticulum contains bezoars. After briefly commenting on the surgical approach, the authors describe two cases in which surgical excision of the diverticulum was followed by improvement.

Western J. Surg., Obst. & Gynecology, Portland, Ore.

61:285-352 (June) 1953

Caudal Anesthesia in Obstetrics with Associated Relaxation Technics. R. N. Rutherford, A. L. Banks, H. M. Lamborn and J. O. Borgen. —p. 285.
Radioactive Colloidal Gold in Carcinoma of Cervix. S. D. Soule.—p. 297.
Anomalies of Stomach and Small Intestine. P. Crabtree.—p. 301.
Combined Physiologic Operation for Peptic Ulcer; (Partial Distal Gastrectomy, Vagotomy and Gastroduodenostomy): Preliminary Report. H. N. Harkins, E. J. Schmitz, H. P. Harper and others.—p. 316.
Lateral Cervical (Branchial) Cysts and Fistulas. S. L. Williams.—p. 319.
Problem of Splenectomy in Diseases of Spleen. II. Splenectomy in "Hypersplenic Syndrome." S. P. Lucia, J. C. Li and M. L. Hunt. —p. 325.
Secondary Amenorrhea: Diagnosed in Charity Obstetrical Clinic. S. M. Martins.—p. 332.
Fertilizing Capacity of Spermatozoa. R. W. Noyes.—p. 342.

Wisconsin Medical Journal, Madison

52:313-372 (June) 1953

Hysteria in Poliomyelitis. M. J. Fox and I. Moskowitz.—p. 313.
Present Concepts of Poliomyelitis: Review of Recent Literature. M. Prouty.—p. 317.
Acute Pneumonia: Appraisal of Six Months' Experience at Veterans Administration Hospital, Wood, Wisconsin. R. Beezy, M. J. Ciccantelli and W. J. Smiles.—p. 322.

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Australian J. Exper. Biol. and M. Science, Adelaide

31:1-104 (Feb.) 1953. Partial Index

Poliomyelitis Virus Pathogenic for Suckling Mice and Monkeys. N. F. Stanley and D. C. Dorman.—p. 1.
*Group A Coxsackie Viruses Isolated from Cases of Poliomyelitis. N. F. Stanley and D. C. Dorman.—p. 9.
Antibodies to Coxsackie Viruses in Pooled Human Serum. N. F. Stanley, D. C. Dorman and J. Ponsford.—p. 17.
Studies on Australian Strains of Coxsackie Virus (Groups A and B). N. F. Stanley, D. C. Dorman and J. Ponsford.—p. 21.
*Hitherto Undescribed Group of Coxsackie Viruses Associated with an Outbreak of Encephalitis. N. F. Stanley, D. C. Dorman and J. Ponsford.—p. 31.
Studies on Intermediary Iron Metabolism. I. Kaldor.—p. 41.
Effect of Foreign Plasma on Capillary Permeability in Unsensitized Animals. B. J. Lake, W. J. Simmonds and A. W. Steinbeck.—p. 55.
Nature of Capillary Damaging Factor in Foreign Plasma. B. J. Lake, W. J. Simmonds and A. W. Steinbeck.—p. 65.

Coxsackie Viruses in Poliomyelitis.—It was demonstrated that Coxsackie viruses were widespread in Sydney during the summer of 1950-1951, when poliomyelitis occurred in epidemic proportions. This paper reports observations on three patients, two paralytic and one nonparalytic, from whom poliomyelitis viruses could not be isolated by monkey inoculation. Coxsackie viruses were isolated from the feces of these three patients. Antibodies to the homologous virus developed in these patients to high titers. One strain of the Coxsackie viruses is related to Dalldorf group A type 4, one to a previously described Australian type (W32) and the third represented a new serological type. It is of interest to determine whether the Coxsackie viruses, isolated from the feces and also responsible for the antibody response, bear any relationship to the disease process or the symptoms (especially paralysis) observed at the time of virus isolation. No attempt has been made to isolate either Coxsackie or poliomyelitis viruses from skeletal muscle of patients, and a Coxsackie virus has not been isolated from human brain or cord post mortem.

Coxsackie Viruses Associated with an Outbreak of Encephalitis.—In the course of epidemiological studies with poliomyelitis and Coxsackie viruses in Australia it became evident that in January, 1952 (mid-summer), a dramatic increase had occurred in the number of cases of encephalitis. This communication describes the isolation and properties of a hitherto undescribed infective agent believed to be closely associated with the outbreak. The laboratory examinations of the outbreak were carried out many weeks after its decline. It has not been proved that the Coxsackie viruses isolated were responsible for the outbreak as none was isolated from central nervous system material. The circumstantial evidence (the isolation of one antigenic type of virus from the feces in a large number of cases and the demonstration of antibodies to the virus in the patient's serum in all cases) is such as to indicate a very close relationship. It is realized, however, that the technique of monkey inoculation for the recovery of poliomyelitis viruses is by no means perfect. As this report includes (with the exception of contacts) only hospitalized patients, an incomplete picture of the outbreak is presented. The outbreak seemed widespread in the Sydney metropolitan area, sufficiently so as to cause comments in the lay press, and a sudden rise in antibody in pooled human serum to the virus thought to be responsible for the outbreak. The demonstration of antibodies to the etiological agent in pooled human serum immediately prior to the time when the disease first became clinically apparent may be explained by the assumption that there was an increase in the number of carriers of the infective agent at this time. The infective agent was shown to be present only in the feces. It was filtrable and had some of the properties of the Coxsackie group of viruses. Because all strains tested produced infection only in suckling mice with resulting lesions in the central nervous system, it was assumed that the agent was a Coxsackie virus. All strains isolated differed slightly from the group B Coxsackie viruses and differed markedly from the group A Coxsackie viruses so far isolated. The differences were sufficient to warrant a temporary designation of Coxsackie group C.

British Medical Journal, London

1:1179-1234 (May 30) 1953

Medicine in Time of Queen Elizabeth the First. A. S. MacNalty.—p. 1179.
Medicine and the Crown. E. A. Underwood.—p. 1185.
Medical and Scientific Exploits of King James IV of Scotland. D. Guthrie.—p. 1191.
Bronchiectasis: Study Centered on Bedford and Its Environs. N. Wynn-Williams.—p. 1194.
Effect of Milk Diet on P. Cynomolgi Infections in Monkeys. R. S. Bray and P. C. C. Garnham.—p. 1200.
Milk Diet, p-Aminobenzoic Acid, and Malaria (P. Berghei): Preliminary Communication. F. Hawking.—p. 1201.
Trial of Goats' Milk in Eczema in Childhood. D. G. Brown and R. L. Holman.—p. 1202.

1:1235-1290 (June 6) 1953

Facts and Fancies About Cancer of Lung. D. W. Smithers.—p. 1235.
Factors in Mortality of Closed Head Injuries. W. Lewin.—p. 1239.
*Streptomycin Resistance in Patients with Pulmonary Tuberculosis Previously Treated with P.A.S. Alone. F. W. A. Turnbull, A. T. Wallace, S. Stewart and J. W. Crofton.—p. 1244.
Treatment of Primary Pulmonary Tuberculosis with P.A.S. R. McL. Todd.—p. 1247.
Incidence of Black Tongue in Antibiotic Treatment. W. Tomaszewski.—p. 1249.
Renal Tumours. R. Shackman and O. Daniel.—p. 1251.
*Peptic Ulcer in Women. D. H. Clark.—p. 1254.
Case of Granuloma Inguinale in Scotland: Response to Aureomycin. A. G. Fergusson and G. B. S. Roberts.—p. 1257.
Pyrimethamine in Treatment of Vivax Malaria: Preliminary Report. J. Singh, A. P. Ray, P. C. Basu and B. G. Misra.—p. 1260.
Goitre and Hypothyroidism Developing During Treatment with P.A.S. H. T. Davies and H.-J. B. Galbraith.—p. 1261.

Streptomycin Resistance in Tuberculosis Patients Treated with *p*-Aminosalicylic Acid.—In the treatment of pulmonary tuberculosis with streptomycin alone the emergence of strains of tubercle bacilli resistant to the drug is well known. That the same thing happens when isoniazid is used alone is now established, but the possibility of resistant strains emerging when pulmonary tuberculosis is treated with sodium or calcium *p*-aminosalicylate alone has received much less attention. This paper revealed that tubercle bacilli from eight of nine patients who had previously received the sodium salt of *p*-aminosalicylic acid alone for pulmonary tuberculosis were found to be resistant to the drug. The original course of treatment lasted from 6 weeks to 16 months, and the interval between the finish of the course and the resistance tests varied from a few days to 39 months. When treated daily with streptomycin and the sodium salt of *p*-aminosalicylic acid, streptomycin-resistant tubercle bacilli emerged in all the eight patients whose bacilli were resistant to the sodium *p*-aminosalicylate before the start of the combined treatment. The streptomycin-resistant bacilli were obtained in six out of the eight cases within three months on combined treatment, in the seventh after four months, and in the eighth after five months. By contrast a slightly streptomycin-resistant organism was isolated after five months' treatment from only one out of six similar cases whose organisms were sensitive to the sodium salt of *p*-aminosalicylic acid at the start of the combined course. Five of the cases had had no previous treatment with the sodium salt of *p*-aminosalicylic acid alone. It is concluded that tubercle bacilli that are resistant to the sodium salt of *p*-aminosalicylic acid are likely to develop if patients with pulmonary tuberculosis are treated with this drug alone; that the resistant organisms may persist for long periods; and that, if the patients are later treated with the sodium salt of *p*-aminosalicylic acid and streptomycin, the sodium salt of *p*-aminosalicylic acid will then no longer prevent or diminish the emergence of streptomycin-resistant bacilli.

Peptic Ulcer in Women.—Many observers assert that symptoms of peptic ulcer and presumably its activity are influenced by menstruation and pregnancy. The evidence presented here was obtained by interviewing 400 women in whom peptic ulcer had been demonstrated. The ulcer was duodenal in 330 cases and gastric in 70. The patients' ages ranged from 21 to 74 years. The symptoms in most cases were of more than five years duration. Often the patient volunteered some information. If not, the standard questions were asked, "Have you noticed any relationship between your ulcer and menstrual period/pregnancy/menopause? Was your ulcer made better or worse,

or did it remain the same?" Apart from paraphrasing these questions according to the patient's understanding of them, no further attempt was made to elaborate a correlation. Of the 400 patients, 291 had had ulcer symptoms before the menopause. In this group 81 noted some relationship between menstruation and ulcer symptoms. In all the symptoms were worse for a few days or a week before the onset of menstruation. In eight the ill-effect persisted during the menstrual period, but none reported increase of ulcer symptoms during the postmenstrual phase. Relief of symptoms occurred in nearly 90% of pregnancies. In most of them the remission occurred early in pregnancy and persisted until term. Those patients who gained no relief had a higher proportion of abnormalities such as albuminuria and hyperemesis. Recurrence of ulcer symptoms was common after parturition especially during the first six months. An unduly large proportion of ulcers start, flare up, or undergo complications at or about the time of the menopause.

Edinburgh Medical Journal

60:249-304 (June) 1953

Treatment of Traumatic Paraplegia in Fractures on Lumbo-Dorsal Spine. I. L. Dick.—p. 249.
Sex-Linked Genes in Man. J. A. F. Roberts.—p. 265.
Case of Amyotrophic Lateral Sclerosis Complicated by Progressive Lipodystrophy. J. Rostowski and J. F. McHarg.—p. 281.
Diabetic Neuropathy. C. H. Davidson.—p. 294.

Harefuah, Jerusalem-Tel Aviv

44:219-242 (May) 1953. **Partial Index**

*Cerebral Hemispherectomy in Cerebral Palsy: Clinical and Electroencephalographic Study. A. J. Belier and M. Streifler.—p. 221.
Myocardial Infarction Due to Coronaritis Following Anti-Diphtheria Serum Injection. Y. Pfeiffer.—p. 225.
*Dangers in Use of Mercurial Diuretics, with Emphasis on Importance of Hypoalbuminemia in Etiology of Complications: Review and Presentation of Case. D. Diengott.—p. 227.

Cerebral Hemispherectomy in Cerebral Palsy.—Hemispherectomy was performed in a 17-year-old girl, who had congenital hemiplegia, frequent epileptic seizures, mental retardation, and aggressive behavior. The girl had been under observation since 1944. She was born after a difficult labor and had generalized convulsive seizures shortly after birth. From the age of 2 years she had had frequent Jacksonian attacks, involving the right half of the body. Anticonvulsive drugs had little effect. Gradually a right spastic hemiplegia developed. In 1945, the left occipital lobe, which harbored a large porencephalic cyst, was excised, but this intervention did not change her seizures, and the left-sided spastic hemiparesis, mental retardation, and aggressive behavior had become severer. Surgical ablation of the left hemisphere was performed in January, 1952, and resulted in complete cessation of the epileptic seizures, alleviation of spasticity in the paretic right extremities, and marked mental improvement. The electroencephalographic records prior to operation showed an area of depressed electrical activity in the left occipital region. There was a zone of impaired electrical activity, displaying irritative features in the left posterior parietal and posterior inferior temporal regions. Hemispherectomy brought about normalization of the electrical activity of the remaining right hemisphere. A short time later, electrical brain rhythms could also be recorded from the left half of the skull. This activity, of low amplitude at the beginning, increased gradually, finally approaching the voltage of activity of the right hemisphere. It is suggested that the sources of this activity are the remaining basal ganglions, particularly the optic thalamus of the extirpated left hemisphere.

Hypoalbuminemia in Complications Resulting from Mercurial Diuretics.—This paper discusses various complications that may result from the use of mercurial diuretics such as loss of minerals, chlorides and water, attacks of gout, renal complications, thromboses, agranulocytosis, and avitaminosis. Reported cases of death after intravenous use of mercurial diuretics are reviewed. Summary of clinical and experimental evidence suggests direct toxic action of the mercury on the myocardium. Four additional cases (two of them fatal) of toxicity due to mercurial diuretics are presented. In these cases toxic manifestations followed immediately after intravenous injection and were apparently caused by direct action on the myocardium. A common factor in all four cases was hypoalbuminemia. Since hypoalbuminemia is noted, or can be inferred, in a large proportion of cases of sudden death after intravenous use of mercurial diuretics, it is suggested that hypoalbuminemia may predispose to such accidents.

Journal of Laryngology and Otology, London

67:301-376 (June) 1953

Rhythmic Palatal Myoclonus with Report of Four Cases. D. L. Chadwick and R. Macbeth.—p. 301.
Myringitis Bullosa Haemorrhagica Its Relationship to Otogenic Encephalitis and Cranial Nerve Paralyses. J. D. K. Dawes.—p. 313.
Modern Developments in Pure Tone Audiometry and Their Application to Clinical Diagnosis and End-Organ Deafness. M. R. Dix and J. D. Hood.—p. 343.
Some Imperfections of Free-Field Voice Tests. P. F. King.—p. 358.

Journal Neurol., Neurosurg. & Psychiatry, London

16:59-126 (May) 1953

Prodromes to Cortical Localization. G. Jefferson.—p. 59.
Studies in Traumatic Epilepsy: 2. Focal Motor and Somatic Sensory Fits: Study of 85 Cases. W. R. Russell and C. W. M. Whitty.—p. 73.
Deposits of Fluorescent Acid-Fast Products in Nervous System and Skeletal Muscles of Adult Rats with Chronic Vitamin-E Deficiency. L. Einarson.—p. 98.
An Extremely Rare Recessive Hereditary Syndrome Including Cerebellar Ataxia, Oligophrenia, Cataract, and Other Features. H. Garland and D. Moorhouse.—p. 110.
Radiological Appearances of Agenesis of Corpus Collosum. P. Sheldon and A. Peyman.—p. 117.

Lancet, London

1:1109-1160 (June 6) 1953

Treatment of Whooping-Cough with Antibiotics: Report by Whooping-Cough Subcommittee of Antibiotics Clinical Trials (Non-Tuberculous Conditions) Committee of Medical Research Council.—p. 1109.
Familial Cretinism. D. Hubble.—p. 1112.
Radioactive-Iodine Studies in Non-Endemic Goitrous Cretinism. E. M. McGirr and J. H. Hutchison.—p. 1117.
*Peripheral Blood Picture After Operation for Portal Hypertension. A. I. S. Macpherson and J. Innes.—p. 1120.
Experimental Electrocardiography of Foetus. C. N. Smyth.—p. 1124.
Erythema Multiforme Exudativum Major (Stevens-Johnson Syndrome). D. M. Davies.—p. 1126.
Stevens-Johnson Syndrome Treated with A.C.T.H. W. G. D. Caldwell.—p. 1127.
Stevens-Johnson Syndrome: Report of Case. P. W. Grant.—p. 1129.

Blood Picture After Operation for Portal Hypertension.—This report is concerned with hematological problems of the syndrome of portal hypertension. Observations in 46 cases of portal hypertension treated either by splenectomy alone or by splenectomy and splenorenal anastomosis indicate that the persistent leukopenia and thrombocytopenia found in this syndrome are rapidly relieved by splenectomy. These findings suggest a relationship between the levels of the leukocytes and platelets in the peripheral blood and enlargement of functional overactivity of the spleen, and is, in accordance with the hematological concept of "hypersplenism." Portacaval anastomosis without splenectomy was done in seven cases of portal hypertension. In three of these, the spleen became smaller from relief of venous congestion, but the pulp hyperplasia persisted, and in no case was the blood picture improved. The effects of ligation of the splenic arterial supply by various operations were observed in five cases. The spleen did not decrease in size after any of these operations. Histological examination of the spleens of three patients in whom the splenic artery had been ligated and divided 15, 18, and 28 months previously, showed either the typical changes of portal hypertension or complete localized infarction. The generalized atrophy of splenic tissue described by other workers was not observed. After splenic arterial ligation no improvement was noted in the blood picture except in one case in which there was extensive infarction of the spleen. Neither relief of portal venous congestion by a portal-systemic venous anastomosis nor reduction in the blood supply

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to the spleen by arterial ligation improves the leukopenia or thrombocytopenia characteristic of portal hypertension. In contrast the changes can be regularly relieved by splenectomy. Hence it may be inferred that the reduction in the leukocytes and platelets is directly related to some influence derived from the cellular components of the enlarged spleen. In no case was there any evidence that any of the operations benefited the red blood cell and hemoglobin levels other than by control and prevention of esophageal bleeding.

Medical Journal of Australia, Sydney

1:609-648 (May 2) 1953. **Partial Index**

The Nervous Child. H. M. North.—p. 612.
Anti-Globulin Sensitization Test as Applied to Brucella Infection: Preliminary Report. A. A. Ferris, W. J. Stevenson and F. A. Lewis.—p. 619.
Hyperplastic Cystic Disease of Breast, with Review of 199 Case Records. J. M. Yeates.—p. 621.
*Role of Nervous Factors in Causation of Peptic Ulceration. B. Robinson.—p. 624.

Causation of Peptic Ulceration.—Robinson traces pathways from various parts of the cortex to the gastric mucosa, suggests mechanisms through which they may affect it, and considers changes that may set these mechanisms into pathological action. Many peptic ulcer patients are outwardly aggressive, ambitious and independent, refusing help and burdening themselves with all kinds of responsibilities. The more they strive for independence and responsibility, the stronger is the unconscious desire to be cared for and to return to the oral stage. Food is then wanted not to satisfy hunger but as a symbol for love and help. This explains also the relief derived from treatment in hospital, as there the patient is able to shed his responsibilities and acquire by way of a seemingly legitimate, socially acceptable compromise, the care, help, attention and security he craves. There seems no doubt that the accepted methods of treatment with rest, sedation, antacids, atropine and diet must be followed during active phases. However, this alone is inadequate. The symptoms must be considered only as indicators of the underlying personality disturbance and continued attention to them will only fix them and contribute to their perpetuation. Time must be spent in the elaboration of the social history and in discovering the patient's attitude towards his environment. Organic nervous lesions are rare in peptic ulceration, but psychological deviations are common and are related, in part at least, to the importance of the alimentary system to the emotional life of the individual. Psychotherapy is a most useful preventive measure during the period of functional symptoms preceding the development of the ulcer and is an essential component of treatment when it is established.

Minerva Chirurgica, Turin

8:181-260 (March 31) 1953. **Partial Index**

Use of Retropneumoperitoneum as Preparation to Interventions on Rectum, Colon, and Retroperitoneal Organs. G. Bertini.—p. 194.
Collateral Circulation After Ligation of Hepatic Artery at Its Point of Origin. A. Bertocchi.—p. 199.
*Pathology and Treatment of Simple, Acute, and Chronic Pneumothorax. L. Biancalana and A. E. Paletto.—p. 209.

Pathology and Treatment of Various Forms of Spontaneous Pneumothorax.—Simple spontaneous pneumothorax appears suddenly in apparently healthy persons, the lesions responsible for it being generally asymptomatic at the time of the onset. This type of pneumothorax has a benign and afebrile course without formation of exudate and usually heals spontaneously. The causative factors of the attack are in most cases ruptured subpleural cysts and localized blebs and bullae. Often the lung expands again; when it does not the condition becomes chronic. The factors which account for the failure of the lung to reexpand are persistence of a bronchial fistula because of a ruptured congenital cyst, intrapleural adhesions, pulmonary and bronchial scar tissue, and often empyema and hemothorax. The acute form of spontaneous pneumothorax is characterized by pain in the chest and a severe dyspnea, making a correct diagnosis easy. The exact cause of spontaneous pneumothorax is best established by thoracoscopy, intrapleural pressure measurements, and bronchography. Bronchoscopy is usually of no diagnostic help. Since cysts and blebs may represent the preparatory phase of a simple spontaneous pneumothorax, their early diagnosis and treatment may prevent the onset of the pneumothorax. Bed rest is the best treatment for simple and acute pneumothorax, but it is ineffectual in chronic cases. The two main types of treatment for chronic pneumothorax are artificial pleurisy produced by irritants introduced into the pleural cavity and surgical procedures. Various types of irritants (including guaiacol, iodoform, iodized oil, dextrose solutions, and silver nitrate) have been placed into the pleural cavity to produce a violent pleurisy accompanied by pleural effusion and fibrin deposition. These close the aperture in the parenchyma, which then expands again. This conservative method is painful and has a severe effect on the patient's general condition, since it causes fever and greatly impairs his respiratory function because of the ensuing fibrothorax. For this reason surgical treatment is preferred. Phrenic paralysis was introduced by Weiss to relax the diaphragm and decrease the tension of the lung and thus permit the pulmonary aperture to heal. The results were not successful. Today chronic pneumothorax is treated either by thoracoscopy or thoracotomy. The latter is the treatment of choice. It is contraindicated only in the presence of bilateral bullae, and in this case induction of pleurisy is the best treatment.

Presse Médicale, Paris

61:787-804 (June 3) 1953

Gynecomastia. L. de Gennes, H. Bricaire and J.-M. Guiot.—p. 787.
*Physiological Correction of Certain Factors of Asthmatic Terrain by Treatment with Pancreatic Extract. D. Santenoise, F. Claude, M. Fontan and M. Roche.—p. 790.
Problem of Diet in Diabetics. Evolution of Physiopathological Concepts. Therapeutic Conclusions. H. Lestradet.—p. 792.
Physiobiological Study of Acclimation Syndrome. A. Cuénot and H. Laborit.—p. 796.
Cutaneous Tuberculin Hypoergy and Anergy. M. Fourestier and A. Blacque-Belair.—p. 799.
*Combined Antibacillary Medications in Treatment of Pulmonary Tuberculosis. E. Bernard, N. Rist and B. Kreis.—p. 802.

Physiological Correction of Asthmatic Terrain.—Patients with asthma have certain functional disturbances predisposing them to acute attacks. These disturbances include an almost constant reduction in the biochemical excitability of the respiratory centers, the very frequent existence of reflex hyperexcitability of the sympathetic centers, especially the vasopulmonary sympathetic centers, and in some cases a reduction in the respiratory capacity of the blood. Excellent therapeutic results have followed the physiological correction of these disturbances with centropneine, a hormone recently isolated from the pancreas. Careful study of 200 patients with asthma treated by this method showed substantial improvement in 161; attacks were either suppressed or reduced in intensity or frequency. The product used in treating these patients, however, was not entirely free from vagotonin, and it was noted that the greater the amount of vagotonin present, the less satisfactory were the results. Further work culminated in the production of a vagotonin-free preparation of centropneine. The effect of this refined preparation was studied in a series of 142 men, women, and children with varying types of asthma. Adults were given from 10 to 30 respiratory units of centropneine before each of three meals; for children, the dose was reduced to from 5 to 10 units. Significant improvement was observed in 125 patients treated for periods of from two weeks to six months; asthmatic attacks, latent oppression, dyspnea on effort, and cough disappeared completely in 40. Several weeks and sometimes months of regular and continuous treatment are usually needed for modification of the asthmatic predisposition. Prolonged treatment presented no difficulty because centropneine, when free from vagotonin, is well tolerated. Treatment was unsuccessful in 17 patients, 7 of whom had severe, inadequately treated respiratory infections. Immoderate use of sympathicomimetic drugs and those de-

pressing the respiratory centers was apparently responsible for failure in eight cases; in two others, predominance of an allergic factor led to partial failure. Supplementary treatment with antibiotics or vaccines and, especially, mineral waters rich in sulphur, may prove highly advantageous.

Combined Antibacterial Medication in Pulmonary Tuberculosis.—The effectiveness of the combined treatment of pulmonary tuberculosis depends primarily on the efficacy of the most active drug in the combination. The belief that the efficacy of one drug is increased by the presence of another is not borne out by either laboratory or clinical tests; the synergistic effect obtained is due to the fact that the supplementary drug affects bacteria that would otherwise be left untouched. The use of drugs in combination is chiefly beneficial because it delays or prevents the development of drug-resistant strains of bacilli, thus making prolonged treatment possible and increasing the chances of cure. These results can only be obtained when the drugs to which the bacilli are sensitive are given together from the beginning of the treatment and in doses that would be effective for each alone; dosage should never be reduced because the drugs are combined. The advantages of the combined treatment are particularly apparent in the highly bacilliferous forms of tuberculosis in which resistance to a single drug develops rapidly.

Schweizerische medizinische Wochenschrift, Basel

83:483-506 (May 23) 1953. **Partial Index**

Pharmacopsychiatry. H. Walter-Büel.—p. 483.
*Problem of Transmission of Hepatitis by Transfusions of Blood and Plasma. A. Hässig, B. von Rütte and K. Vettiger.—p. 487.
Introduction of New Index and Pediometer into Pediatric Practice. S. Simkó.—p. 492.
Phenylbutazone (Butazolidin) in Treatment of Rheumatic Diseases. K. Huber and D. Binswanger.—p. 496.

Transmission of Hepatitis by Blood and Plasma Transfusions.—Of 189 patients who were given transfusions of whole blood in 1950 and 1951, only 1 had icterus within six months after the transfusion. The risk of hepatitis following infusion of whole blood is small and is probably far below 1%. The blood of seven donors who had jaundice one to five years before was tranfused to eight recipients. The blood of additional 77 donors who had had jaundice five or more years before they donated blood was transfused to 92 recipients. None of these 100 recipients had hepatitis within six months after the transfusion. Although the number is too small for definite conclusions, these figures suggest that convalescent carriers are of little importance in transmission of hepatitis. The management of the blood donor service of the Swiss Red Cross decreed in 1952 that any person who had once had jaundice should be excluded from its donor service. Although this resolution means an average 8% loss of blood donors, the authors agree with it because it is well established that some persons are carriers of hepatitis virus for several years and the question of the average duration of carrying convalescent virus has not been answered. If for some reason it should not be possible to exclude donors who once had had jaundice, their blood should be used only for single transfusions but not for the production of a pooled plasma. Of 189 patients admitted to the City Hospital of Basel who had been given 362 transfusions of dried plasma and 516 transfusions of whole blood, 8 had inoculation hepatitis for which the dried plasma was held responsible. The incidence of hepatitis with respect to the number of recipients was 4.2%, and with respect to the number of administered dried plasma units 2.2%. Of 90 recipients of 135 dried plasma units who did not receive whole blood, 4 had epidemiologically established inoculation hepatitis caused by dried plasma. The incidence of hepatitis with respect to the number of recipients, was 4.4% and with respect to the number of administered dried plasma units 2.9%. Of 126 plasma pools, 4 were definitely icterogenic and 2 others possibly were. Since none of the donors who had contributed to the plasma pools had had icterus, it may be assumed that healthy carriers rather than incubation virus carriers played the most important part in the contamination of the plasma pools with hepatitis virus. The conclusion for the practitioner is that pooled plasma should be employed only in cases in which because of the gravity of the patient's condition the risk of late damage caused by inoculation hepatitis appears to be insignificant as compared to the expected therapeutic usefulness of the pooled plasma.

Semaine des Hôpitaux de Paris

29:1533-1586 (May 10) 1953

Significance of Electrophoresis in Study of Subacute Infectious Endocarditis. E. Donzelot, H. Kaufmann, J. M. le Bozec and S. de Mende.—p. 1533.
*Clinical and Prognostic Value of Electrocardiography in Treatment of Subacute Infectious Endocarditis. E. Donzelot, J. M. le Bozec, H. Kaufmann and J. Laham.—p. 1540.
Immediate and Long-Term Prognosis of Subacute Infectious Endocarditis. E. Donzelot, H. Kaufmann, J. M. le Bozec and J. E. Escalle.—p. 1544.
*Clinical and Physiopathological Considerations in Treatment of Angina Pectoris with Heparin. E. Donzelot, H. Kaufmann and J. M. le Bozec.—p. 1549.

Electrocardiography in Subacute Infectious Endocarditis.—Electrocardiograms made routinely before, during, and after treatment in a series of 200 patients with subacute infectious endocarditis were studied to determine their diagnostic and prognostic value. A diagnosis of subacute infectious endocarditis was rarely established when auricular fibrillation or acquired valvular heart disease affecting the right side of the heart was reflected in the electrocardiograms; acquired heart disease affecting the left side of the heart, on the other hand, was frequently complicated by subacute infectious endocarditis. Signs of acute cor pulmonale and myocardial impairment appearing in electrocardiograms made during treatment often presaged irreversible heart failure. Electrocardiographic changes are most pronounced in resistant cases and those in which treatment is late. The abacterial forms of the disease show a preponderance of changes; in electrocardiograms made before treatment was started, cardiac insufficiency was found in 15% of the patients with negative blood cultures as compared with 4.5% of those with positive blood cultures, while during treatment heart failure occurred in 22% of the patients with negative cultures and in only 11% of the others. Bundle-branch block and coronary impairment predominate in patients with aortic involvement, possibly because the severe, primary forms of subacute infectious endocarditis are almost always localized in the aortic valves. The principal changes in patients with mitral involvement are those indicative of acute cor pulmonale and, later, auricular fibrillation. Fear of possible complications must not be allowed to interfere with the use of penicillin, which offers the only hope of cure.

Treatment of Angina with Heparin.—Study of 53 patients, most of whom have been followed for a year, shows that attacks of pain can be suppressed by the intravenous injection of a very small dose of heparin (e. g., 25 mg.) in about 70% of patients with angina pectoris. Improvement was secured in nearly 100% of those with typical angina occurring on effort and relieved within about 5 minutes by glyceryl trinitrate; the results in patients with bundle-branch block or a sequela to myocardial infarction whose chest pain was not clearly connected with effort or clearly relieved by glyceryl trinitrate were less satisfactory. Patients who respond promptly to glyceryl trinitrate are almost always benefited by heparin; when glyceryl trinitrate is ineffective, heparin treatment is not likely to be successful. The same patients benefit by both remedies, but glyceryl trinitrate is an emergency treatment to be used during an anginal attack, while heparin is a basic treatment applicable to the anginal state. The efficacy of heparin is greatest when the anginal attacks are clearly due to effort; patients in whom they are related to emotion also prove responsive to its action. Atypical pain accompanied by vasomotor phenomena of the kind found mostly in hypertensive patients is not affected by either heparin or glyceryl trinitrate. Three patients with calcified aortic stenosis who had for a long time had frequent attacks and who required

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several glyceryl trinitrate tablets daily were benefited by heparin to such an extent that within the first days of treatment glyceryl trinitrate became unnecessary. The effectiveness of heparin treatment does not seem to be dependent on the blood picture; excellent results were obtained in a patient in whom hypocoagulability was pronounced, and sustained relief of anginal attacks were secured in another patient with persistent hypercoagulability. Patients receiving heparin must follow the usual tranquil regimen, even in the absence of attacks; although their tolerance for exertion is increased, a severe attack may be produced by any unusual effort. Improvement is usually constant as long as the injections are continued, but anginal attacks recur when treatment is suspended even after a period of several months. Failure of heparin to produce good results within three weeks indicates that the dose should be changed; addition of another drug, such as ethyl biscoumacetate (Tromexan ethyl acetate), may also prove advantageous. Laboratory studies showing that the effects of heparin injection are antagonistic to those of sympathetic excitation suggest that the action of heparin, though exerted at a different level, is similar to that of the sympathicolytics. Heparin apparently influences the humoral effects of nervous excitation, while the sympathicolytics tend to suppress the excitation itself.

Ugeskrift for Laeger, Copenhagen

115:627-670 (April 23) 1953. **Partial Index**

*Tumors of Small Intestine. M. Faber, A. Gjersøe and G. Munk-Andersen.—p. 627.
Metastasis from Cancer of Thyroid Simulating Lateral Aberrant Tumor of Thyroid. A. Birch-Jensen and J. B. Dalgaard.—p. 635.
Relaxing Effect of Mephenesin on Psychogenic Tension. K. Nymgård. —p. 641.
Fate of Patients After Attempted Suicide. After-Examination of 500 Patients. H. Hove.—p. 645.

Tumors of Small Intestine.—Faber and his associates described 45 cases of tumor of the small intestine in patients aged between 40 and 70. Fifteen tumors were benign, 10 were sarcomas, 9 of which were apparently primary in the small intestine and one presumably metastatic, and 20 were carcinomas. While the benign tumors and sarcomas were usually distributed throughout the small intestine, the carcinomas showed a marked tendency to occur at the ends of the intestine. In 12 cases there were no symptoms until an acute abdominal catastrophe occurred, in 11 an acute abdominal crisis occurred after symptoms of some duration, in 12 the symptoms were those generally seen in tumors elsewhere in the gastrointestinal tract, and in 9 cases the only sign was bleeding, either typical melena or occult with anemia. The prognosis for tumors of the small intestine has greatly improved in recent years. Careful examination including vaginal exploration and roentgen examination of the small intestine as a rule gives the diagnosis. If the results were negative and particularly if occult bleeding continues, exploratory laparotomy is necessary.

Wiener klinische Wochenschrift, Vienna

65:369-452 (May 15) 1953. **Partial Index**

Exceptional Case of Colpocleisis. T. Antoine.—p. 372.
Symptomatology and Treatment of Intestinal Parasites. R. Boller.—p. 373.
Thoracoplasty or Pneumonolysis. A. Kenner.—p. 376.
Radical and Palliative Excision of Gastrointestinal Carcinomas. A. Plenk.—p. 384.
*Report on Results of Treatment of Carcinoma of the Breast at the Central Roentgen Institute of the Vienna-Lainz Hospital Between 1937 and 1947. W. Heilig.—p. 399.
*Irradiation Therapy of Carcinoma of Uterine Cervix. E. Maier.—p. 412.

Irradiation Therapy of Carcinoma of the Breast.—Between 1937 and 1947, 580 women and 2 men with carcinoma of the breast were given irradiation treatment at the central roentgen institute of the Vienna-Lainz Hospital. Of these, 234 with stage 1 to 3 carcinoma, according to Steinthal's classification, were given intensive local irradiation between 1937 and 1945 (first period of treatment), 273 with stage 4 carcinoma (generalized carcinosis) were given small dose-irradiation treatment of the entire body between 1938 and 1947 (second period of treatment), and 75 patients with stage 1 to 3 carcinoma received combined intensive local irradiation and prophylactic irradiation of the entire body between 1945 and 1947 (third period of treatment). In patients with carcinoma of stage 1 to 3 treated during the first period, the technique of fractionated local irradiation consisted of the usual three field-irradiation with 175 kv., 6 ma., 0.5 mm. Cu plus 1 mm. aluminum filter at a distance of 40 cm., a field dose of 240 r, each field repeated seven times, one field daily, with a total serial dose of 5040 r surface. Irradiation treatment was instituted on the third postoperative day. The local series were not repeated. Of the 234 patients of the first irradiation period 117 (50%) obtained a five year cure. In the 273 patients with stage 4 carcinoma treated during the second period, a permanent cure could not be expected as a result of the irradiation therapy; despite that, an average survival time up to two years with working capacity maintained in the complete or nearly complete absence of pain, was obtained by entire body-irradiation with alternating posterior and anterior entire body-fields, 175 kv. at the tube, 4 ma., 0.5 mm. Cu plus 1 mm. aluminum filter, at a distance of 150 cm., with a dose of 24 r per single session. A series consisted of 10 single sessions. The interval between two series varied from three months to four weeks according to tolerance. Of the 75 patients treated during the third treatment period, 39 (52%) obtained a five year cure. This increased ratio of five year recoveries was obtained by the combined treatment with local irradiation (seven times 240 r) and an entire body-irradiation series of 10 times 24 r with alternating anterior and posterior fields two months after the last local irradiation and repeated twice at six week intervals.

Irradiation Treatment of Cancer of Uterine Cervix.—Between 1937 and 1946, 1,410 women with carcinoma of the uterine cervix were examined at the irradiation department of the Vienna-Lainz hospital. Only 1,048 received complete irradiation treatment, while the large number of 362 patients did not, because of the political events and the war occurring during the reported period. Of the 1,410 patients, 56 had stage 1 carcinoma of the cervix uteri according to the classification of the 1937 committee of the League of Nations, 139 stage 2, 875 stage 3, and 340 stage 4. Treatment consisted of near-radium irradiation and radium irradiation at a distance of 6 or 10 cm., depending on the location of the tumor. The near-radium irradiation was carried out according to Forssell's principle, with capsule-shaped vaginal applicators equipped with 80 mg. radium element and with an intrauterine probe equipped with 40 mg. radium element. This treatment was practiced in three stages, i. e., vaginal application for 30 hours, repeated for 24 hours after an interval of 14 days, and followed by treatment with the intrauterine probe for 72 hours after a second interval of 14 days. Radium treatment at a distance of 6 cm. was given with 6.93 r per minute at the skin surface and at a distance of 10 cm. with 3.4 r per minute. Four field-irradiation was used; one field received on the average at the distance of 6 cm. a total dose of 12,500 r in 30 hours. Half of the surface dose was obtained with the 6 cm. distance and a 3 cm. depth or with a distance of 10 cm. and a 4 cm. depth. One field was irradiated daily for two hours, so that the total duration of treatment varied from six to eight weeks, and the lateral parametriums received a total dose of approximately 2,000 r. Of the 1,048 patients who have completed their treatment, 276 were symptom-free for five or more years, i. e., a relative average recovery rate of 39.2% in the four stages of the disease. This recovery rate was somewhat smaller than that of 41.7% obtained during the period of 1935 to 1938; the less favorable results were due to the war and undernourishment. The results obtained with irradiation therapy for carcinoma of uterine cervix at the Vienna-Lainz hospital correspond with those obtained at other large irradiation institutes according to Heymann's annual report of 1951. Satisfactory results did not depend only on treatment but also on the periodic control of the patients once treated and on the general care they had received. The combination of all favorable factors promise a successful campaign against carcinoma of the uterine cervix.

BOOK REVIEWS

Atlas of Medical Mycology. By Emma Sadler Moss, B.S., B.M., M.D., Director, Department of Pathology, Charity Hospital of Louisiana at New Orleans, and Albert Louis McQuown, B.S., B.M., M.D., Pathologist, Our Lady of Lake Sanitarium, Baton Rouge, Louisiana. Cloth. $8. Pp. 245, with 248 illustrations. Williams & Wilkins Company, Mount Royal and Guilford Aves., Baltimore 2, 1953.

This book does not constitute a major contribution to the literature of medical mycology. It does, however, constitute, particularly for the uninitiated, a useful volume, because of the number of pictures included and, in some instances, their high caliber. The text of the book appears to have been drawn from other more lengthy texts or manuals in the field and actually constitutes a synopsis. The subject is dealt with in terms of the diseases that fungi produce; consequently there is little continuity or over-all plan. A more constructive approach would have been an adequate verbal consideration of the field joined with the excellent pictures that are presented. The field is at present in need of an adequate reference of this type, but the present book does not answer this need. The subject immunology of fungi could bear much more extensive treatment, as could the basic biology of the organisms themselves. The book does seem to be adequate, although far from complete, in considering the clinical aspects of the individual diseases presented.

History of Anesthesia: With Emphasis on the Nurse Specialist. By Virginia S. Thatcher. Cloth. $5. Pp. 289, with 22 illustrations. J. B. Lippincott Company, 227-231 S. 6th St., Philadelphia 5; Aldine House, 10-13 Bedford St., London, W.C.2; 2083 Guy St., Montreal, 1953.

In this excellent reference book, the author emphasizes the position nurses have attained in the conquest of pain and presents arguments as to the philosophical, historical, anthropological, and legal basis for nurses in the field of anesthesia. Little reference is made to the history of anesthesia other than its application to nurses. Special emphasis is placed on the neglect of anesthesia as an art and science in the latter half of the 19th and early first quarter of the 20th centuries, with the duties of the anesthetist being assigned to the lowest ranking doctor. Eventually, nurses were pressed into service and handled anesthesia administration commendably because of their greater interest in the work. More attention is given to the development of the art of anesthesia than to the discovery of agents and techniques. The second half of the book deals with the story of the American Association of Nurse Anesthetists and its founders.

There has been a liberal use of footnotes and an index is appended, both of which will make this work valuable as a reference to any one interested in the role nurses have played in the history of anesthesia. The author seems most enthusiastic about her subject and has presented a convincing argument for the continued presence of nurses in the field of anesthesia administration.

Wiederherstellungschirurgie und Traumatologie. Jahrbuch [1]. Redactor: M. Lange. Editores: A. de Araujo, et al. [In German, English and French.] Cloth. 36 Swiss francs. Pp. 262, with illustrations. S. Karger, Holbeinstrasse 22, Basel, 1953.

This is an annual survey of reconstructive surgery and traumatology that records progress in the field. Each subject is discussed in German, English, and French. The various topics are presented by authorities who project their own views and personal experiences. When opinions of these surgeons differ widely a member of the editorial board has appended a critical comment. During the last 10 years surgeons in many different countries have made important contributions in reconstructive surgery of the locomotor system; many of these are included. Emphasis has been placed on the value and limits of intramedullary pinning, aseptic necrosis of the femoral head after nailing, and treatment of irreparable nerve palsy. The following discussions deserve special commendation: Intramedullary Pinning, Complication, and Technique by F. H. Vom Saal; Traitement des nécroses traumatiques de la tête du fémur by R. Merle d'Aubigné; Reconstruction Operations for Ulnar Paralysis when the Nerve is Irreparable by S. Bunnell; Die Behandlung der poliomyelitischen Lähmung des Daumens by A. Steindler; and Median and Ulnar Nerve Injuries by J. W. Littler. This international yearbook is a step in the right direction, since it presents a cooperative effort on the part of leading surgeons in different countries. The trilingual approach should make it valuable to those who are deficient in one or two of the languages included.

Your Arthritis: What You Can Do About It. By Alfred E. Phelps, M.D. Second edition. Cloth. $3. Pp. 192, with illustrations by James Macdonald. William Morrow & Company, Inc., 425 Fourth Ave., New York 16, 1953.

This small volume has been revised to include recent developments in the field of rheumatology. The book was written for the lay person with arthritis, to help him to understand better the nature of this disease and to aid him to appreciate the problems involved in his treatment. It covers briefly the historical background of arthritis, prevalence of the disease, anatomy of articular structures, treatment of various types of arthritis, and prevention of deformities. There is a discussion of corticotropin, cortisone, and hydrocortisone, and a detailed review of their discovery. The book is printed in large, clear type and has a number of anatomic illustrations. The style is easy to follow.

The author fails, however, to achieve his objective in that the illustrations and text are far too technical for the average lay person. The book is too elementary to be used by physicians or medical students and is too academic to be of any help to the arthritic patient in understanding his ailment. It is difficult to see how a person with any type of arthritis would benefit from a detailed account of the discovery of the hormonal drugs or from an involved discussion of the acid-alkali balance in the blood. The subject of rheumatology is briefly and superficially covered in this volume, but it is doubtful if the arthritic patient would learn how to live with his disease from reading it.

Textbook of Public Health (Formerly Hope and Stallybrass). By W. M. Frazer, O. B.E., M.D., Ch.B., Medical Officer of Health, City and Port of Liverpool, England. Thirteenth edition. Cloth. $8.50. Pp. 663, with illustrations. [Williams & Wilkins Company, Mount Royal and Guilford Aves., Baltimore 2]; E. & S. Livingstone, 16 and 17 Teviot Pl., Edinburgh 1, 1953.

This is the latest edition of a book that has become a standard textbook and manual for public health practice in England. The volume covers the knowledge required for the Certificate and Diploma in Public Health under the rules of the General Medical Council. Besides being a valuable compendium of technical knowledge, which has been brought up-to-date by extensive revision, the work is notable for its excellent chapters dealing with administrative and governmental problems of health protection and medical care.

When the previous edition appeared in 1948 the National Health Service Act was so recent that little could be included other than the basic legislation. The intervening years have seen the development of administrative machinery for carrying out the act, modification of the basic law, and passage of the National Assistance Act. Dr. Frazer presents a concise description of these and their effect on public health and medical practice. In an open-minded manner, he intersperses highly objective appraisals, finding both good and bad in the new legislation. The reader who seeks technical information on public hygiene as well as critical factual descriptions of the effect of recent British social legislation on health and medical matters will welcome this new edition.

These book reviews have been prepared by competent authorities but do not represent the opinions of any official bodies unless specifically so stated.

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QUERIES AND MINOR NOTES

DRUGS TO AVOID IN DIABETICS

To the Editor:—*It is often stated in medical books and literature that certain commonly used drugs may be used except in diabetics. Please list some of the common drugs and their effects on diabetics.*

R. J. Turner, M.D., Sheridan, Ark.

Answer.—Few drugs must be denied to diabetics, but a great many should be used with caution. 1. Elixirs, syrups, and similar preparations with sugar for flavoring are undesirable for a diabetic. 2. Bromides and iodides may lead to pustular eruptions in diabetics, as well as in normal persons. In diabetics, however, any inflammatory lesion is deleterious and lowers the efficacy of insulin. 3. Extracts of the pituitary, thyroid, or adrenals are harmful to diabetics, because they increase the severity of diabetes and counteract the activity of insulin. Epinephrine (Adrenalin) and vasopressin (Pitressin) may be valuable aids in treating insulin hypoglycemia. Cortisone leads to hyperglycemia and glycosuria, but as yet the effects reported have always been temporary. 4. Opium is contraindicated in diabetic coma. Opium so depresses the labored Kussmaul respiration that death in a few moments may follow even a small dose. For this same reason, surgeons avoid the usual doses of morphine before operation on diabetics and are cautious about its use even in small doses. This should be considered particularly when ether is used as an anesthetic, because, through the depressing effect of morphine on the respiration, a larger quantity of ether would be required and ether exerts a toxic action on the glycogen-depleted liver and tends to increase an existing acidosis. Overdosage with tribromoethanol (Avertin), scopolamine, barbiturates, and paraldehyde should be avoided. 5. Iron is contraindicated in hemochromatosis, which so commonly leads to diabetes, and this holds even though anemia appears extreme. 6. Bishydroxycoumarin (Dicumarol) in a diabetic should be used with especial care because of the increased frequency of vascular disease that increases the frequency of hemorrhage in a diabetic. 7. Preparations of salicylic acid are not harmful to the diabetic, but, unless one is alert, the confusing reaction in the urine, when testing for diacetic acid with the ferric chloride test, may be misinterpreted and acidosis wrongly diagnosed. In one such instance, a person was sent home from abroad to avoid diabetic coma because of the confusion resulting from the salicylic acid coating of a pill commonly used for constipation.

TREATMENT OF RHEUMATOID SPONDYLITIS

To the Editor:—*Please describe the latest treatment for Marie-Strümpell disease.*

O. S. Goodwin, M.D., Apex, N. C.

Answer.—The recognized treatment for rheumatoid spondylitis (Marie-Strümpell disease) is roentgen irradiation in conjunction with rest, heat, exercises, and immobilization. This treatment results in lessened pain and stiffness and increased motion in 80% of patients with rheumatoid spondylitis. There is a decrease in the erythrocyte sedimentation rate, but the abnormal roentgen findings remain unaltered and may even increase.

The factors necessary for the roentgen treatment are as follows: 140 kv. with 0.5 mm. of copper, 1 mm. of aluminum filtration, a half value layer of 0.6 mm. copper, 50 cm. skin target distance, and 150 r (measured in air) to each part per treatment, and a total of 600 r for the first course. The ports selected are the sacroiliac (12 by 20 cm.), lumbar spine (20 by 8 cm.), dorsal spine (20 by 8 cm.), and the cervical spine (12 by 20 cm.).

The sacroiliac and the lumbar regions are treated as a unit. The sacroiliac is treated in the morning and the lumbar region in the afternoon daily for four successive days. The dorsal and the cervical regions are treated during the next four days, with one port exposed in the morning and the other port in the afternoon. A second course may be repeated in two months, with the painful sites as ports and 150 r per day given for three successive days, or a total of 450 r. The second course may be repeated if pain persists.

Additional measures are rest in bed with a nonsagging firm mattress until pain, tenderness, and muscle spasm have subsided. The spine should be kept hyperextended to minimize the strain of the spinal ligaments and to obtain optimum chest expansion. This is usually achieved by removing the pillows from under the head. If this procedure fails, a plastic jacket is indicated. Deep breathing exercises are extremely important in order to prevent the costovertebral joint from becoming fixed. Forced deep breathing should be practiced by the patient several times daily for five minute periods. Heat in the form most comforting to the patient should be applied three or four times daily.

VITAMIN B_{12}

To the Editor:—*Does vitamin B_{12} have any toxic effect when given in doses as large as 1 mg. daily? Is there any indication for the use of massive doses of vitamin B_{12} in paralysis agitans (Parkinson's disease)?* *M.D., Kentucky.*

Answer.—High potency solutions of vitamin B_{12} for injection have been available for investigational use for two and one-half years and were approved for sale by the Food and Drug Administration in September, 1952. Daily doses of 1,000 μg or more have been administered to numerous patients for periods of several months. To-date, there have been no toxic manifestations or any specific contraindications to its use. In view of the effectiveness of this material in the treatment of macrocytic anemias, the question has frequently been raised as to whether large doses directly affect the hematopoietic system. The weight of research evidence points to vitamin B_{12} as the essential factor in the maturation of erythrocytes and does not indicate that it plays any role in their production. Hematological studies of many patients treated with large doses have failed to show any increase in the total erythrocyte count. There is no published information regarding the use of large doses of vitamin B_{12} in paralysis agitans, and there is nothing in the clinical data thus far accumulated to suggest that it would be of any specific benefit. Certain general effects, such as a sense of well-being, may be expected from its use.

HISTOLOGICAL SECTIONS

To the Editor:—*A pathologist here delays histological examination of biopsy material as long as seven days. Is there a shorter procedure? Please give details.*

M.D., Dominican Republic.

Answer.—If an autotechnicon is used, the tissues can be fixed, dehydrated, and ready for embedding in paraffin in 18 hours. The sections can be cut the day after they are received and stained the next day, then they are ready for diagnosis. Although the period varies, depending on the number of specimens, 48 hours are required, including the typing of reports. If an autotechnicon is not available, the process takes longer. The tissue must be properly fixed, which may take a whole night, depending on the size of the specimen. The tissue is then put in acetone, which is changed every half hour for three changes, then in xylene for 15 minutes, and then in two changes of paraffin, in the first usually for 20 minutes and in

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the second for one hour. Again, however, the time required depends on the size of the specimen. After the tissue is embedded and cut, the slides can be dried in an oven for several hours, stained, and interpreted the same day.

NODULES IN TENDONS OF HANDS

To the Editor:—*A patient, 52 years of age, has a nodule tendonitis involving the palm of the right hand over the head of the fifth metacarpal bone. An orthopedic surgeon advised that no surgery be done at this time, unless a trigger finger develops. Is x-ray therapy of value in this type of trauma?* *Leonard B. Fauquier, M.D., Jersey City, N. J.*

Answer.—The occurrence of nodules in the tendons of the hand is fairly common, especially during an attack of chronic rheumatoid arthritis. They are not usually treated surgically, unless a painful trigger finger results. X-ray therapy is often helpful.

REMOVAL OF PERMANGANATE STAINS

To the Editor:—*What will remove potassium permanganate stains from glass urethral syringes?*

Charles H. Codelle, M.D., New York.

Answer.—The brown stains result from manganese oxide deposits formed when potassium permanganate comes in contact with organic matter. They may be removed by soaking of the syringe in a strong mineral acid (sulfuric or hydrochloric) or in a fresh solution of nitrous acid prepared by the addition of sulfuric acid to a nitrite in a cold atmosphere. Care must be exercised to prevent inhalation or exposure of the eyes and skin to the highly toxic fumes. The acid solutions are extremely corrosive and will remove the enamel from graduations on the syringes.

SYSTOLIC "CLICKS"

To the Editor:—*I have observed for the past two years a 35-year-old man who has sharp precordial pain that is unrelated to effort and that lasts less than a second. Electrocardiograms, chest roentgenograms, and results of cardiac examination have always been normal. The only unusual thing has been the presence of a clicking (not split) second heart sound, heard over the precordium but loudest in the mitral area. Please explain the significance of this sound.*

Harold L. Scales, M.D., Teaneck, N. J.

Answer.—The second heart sound is produced by closure of the aortic and pulmonary valves and is, therefore, loudest at the base of the heart. The origin of systolic "clicks" is uncertain but is probably extracardiac. The sounds are often noted in patients with normal hearts. They are always louder at or are confined to the apex and are most clearly heard when the patient is recumbent. The patient in question probably has such an extracardiac sound late in systole, which is loud enough to mask the second sound. This has no pathological significance and cannot be related to the patient's symptom of chest pain.

CEREBRAL COMPLICATIONS OF RUBELLA

To the Editor:—*I have encountered a case of rubella (not rubeola) encephalitis. What is the proper treatment? Is any antibiotic of value?*

K. Clippinger, M.D., Covington, Ohio.

Answer.—There is no specific treatment for the cerebral complications of rubella (German measles). All treatment is symptomatic. Strict bed rest is important throughout the acute stage of the illness. Chloral hydrate, paraldehyde, or intramuscularly administered phenobarbital sodium can be used for the restlessness. The use of opiates should be avoided. If urinary retention or incontinence is present, an indwelling catheter should be employed, with small doses of sulfonamides. Adequate nourishment must be maintained. If the patient is comatose, glucose and amino acids should be given intravenously; if not in deep coma, tube feeding may be employed. Recovery is usually fairly rapid and complete. There are no new drugs or antibiotics that are helpful in this condition.

PENICILLIN REACTIONS

To the Editor:—*Will the addition of an antihistaminic to parenterally administered penicillin control the occurrence of sensitivity reactions due to the penicillin?*

M.D., California.

Answer.—The addition of an antihistaminic drug to penicillin for parenteral administration will not prevent the occurrence of sensitivity reactions. Administration of antihistaminic drugs after the development of skin rashes due to penicillin sensitization will decrease, sometimes markedly, the pruritis that develops, but the other manifestations of the reaction, such as rash or joint pains, fever, and lymphadenopathy in the severer cases are not affected to any appreciable degree by the drug. No other substances are known that will decrease the reactivity to penicillin in patients in whom a sensitivity to this agent has developed. It is important to point out that not all such sensitive persons will react every time they receive penicillin; in some instances, readministration of the drug to a previously sensitive person will not provoke a second allergic reaction. In some patients, not all, sensitivity to penicillin G is not accompanied by allergy to penicillin O; in these cases the latter agent can be given if there is fear of a dangerous reaction to penicillin G. It is possible, however, for patients to become sensitive to penicillin O, and some persons are allergic to both types of the antibiotic.

ASTHMA AND ODORS OF PAINT

To the Editor:—*A man, 30, recently had his first attack of bronchial asthma. He works in a paint and varnish factory as a shipping clerk. There is no history of asthma. He had rheumatic fever at age 11. Physical examination reveals a boggy nasal mucosa and tearing of the eyes. Examination of the heart was within normal limits. The lungs appear normal except during the asthmatic attacks. The blood pressure is 130/70 mm. Hg. A blood cell count revealed eosinophilia, 6%. The patient responded to antiallergic medication, and since he has stopped working his asthma has improved. Could you suggest diagnostic procedures that would aid in establishing the allergen precipitating this patient's bronchial asthma?*

E. Edward Benzier, M.D., Rego Park, N. Y.

Answer.—The odors of paint and varnish frequently incite attacks of asthma. In the majority of such instances they act as irritating vapors that can produce an attack in the presence of a separate allergic factor. Thus they behave like coal smoke, gasoline fumes, and other irritating gases. However, it is also possible that the patient is exposed in his occupation to specific allergy-producing substances. Among such possible causes are linseed products. These allergens as well as others should be employed in testing this patient. The safest procedure would be the scratch test.

SIMULTANEOUS ADMINISTRATION

To the Editor:—*In the Queries and Minor Notes, in "Simultaneous Administration" (The Journal, May 16, 1953, page 296), a subscriber raised the question of the safety of administering 5% glucose in sodium chloride and citrated whole blood simultaneously. The answer given was that this procedure was safe provided the solutions were isotonic. I should like to suggest that this answer is incomplete. The effect of exposure of human red blood cells to 5% dextrose is immediate swelling of the cells because of imbibition of water. This can be of sufficient magnitude to cause varying degrees of hemolysis. This effect is more pronounced in stored blood than in fresh blood, since a constant characteristic of refrigerated red blood cells is a decrease in osmotic resistance. The type of procedure mentioned should be reserved for extreme emergencies, since it will undoubtedly lead to varying degrees of intravascular hemolysis of the transfused cells.*

John G. Gibson II, M.D.
Research Associate in Medicine
Harvard Medical School
25 Shattuck St.
Boston 15.